



Universitas Kristen Indonesia

Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 061/UKI.F5.D/HKP.3.5.6/2021
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

- MENIMBANG** : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendid di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk member Kuliah Pakar
- MENGINGAT** : 1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KBK FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 teni pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

- MENETAPKAN** : 1. Penugasan dalam memberikan Kuliah Pakar :
- | | |
|--------------|-------------------------------|
| Nama | dr. Fajar L. Gultom, Sp.PA |
| Departemen | Patologi Anatomi |
| Blok | 5 (Biomedik) |
| Judul Materi | Pendahuluan Patologi Anatomik |
| Semester | genap 2020/2021 |
| Kelas | A : 0,21 SKS
B : 0,21 SKS |
| SKS | 0,42 SKS |
2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputu ini akan diperbaiki sebagaimana mestinya

Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 15 April 2021

Dekan,

Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:

1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI

● RENDAH HATI ● BERBAGI DAN PEDULI ● PROFESIONAL ● BERTANGGUNG JAWAB ● DISI



Pathology Introduction

Fajar L. Gultom

Departemen Patologi Anatomik FK UKI

April 2021

Pathology

- Pathos: Suffering
- Logos: Study
- **Pathology: Study of Disease**
- **“All diseases are alteration of normal histology” - Virchow –**
Father of Modern Pathology

Pathology

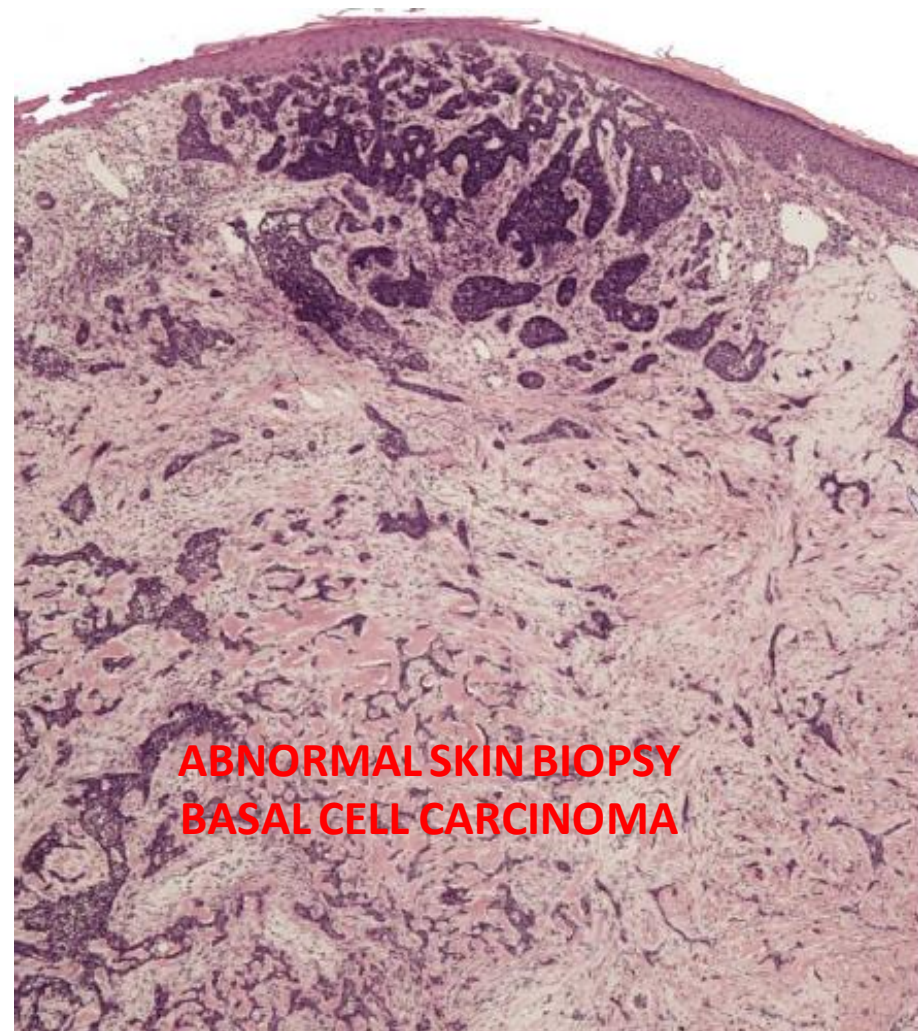
Using your **basic Histology** for
understanding disease

Histology → Cellular Pathology

Normal → Abnormal

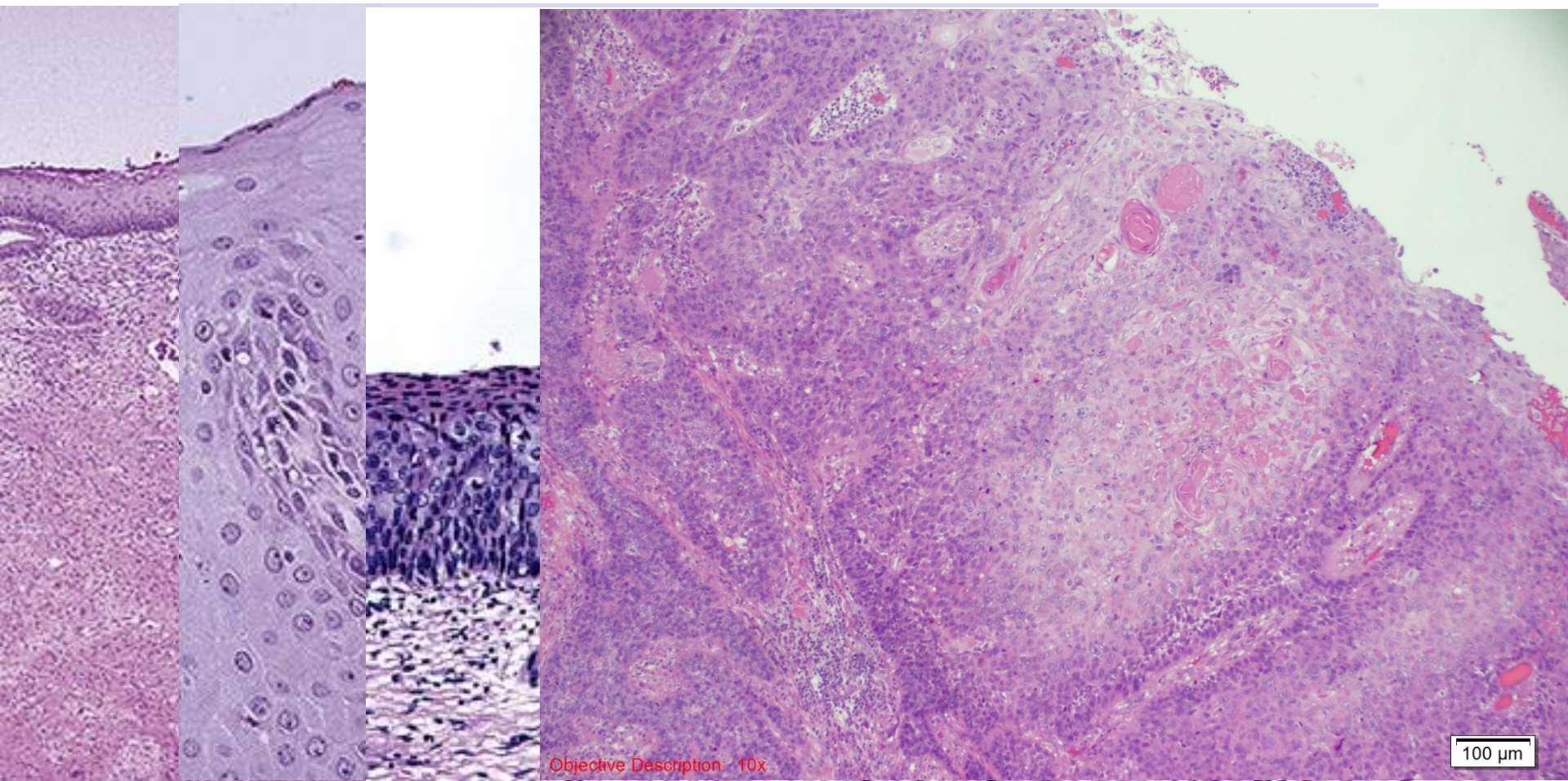


NORMAL SKIN BIOPSY



**ABNORMAL SKIN BIOPSY
BASAL CELL CARCINOMA**

Pathology



Normal cervix biopsy

Cervicitis

Dysplasia

Cancer

Basic Concepts

Morphology Classification of Human Disease:

- 1. Degenerative**
- 2. Inflammation**
- 3. Neoplasma**

Classification

Wikipedia..

- **Cause**
- **Symptoms**
- **Pathogenesis**
- **Organ systems**

Complicated..??

1. Degenerative

Degenerative disease:

“Result of continuous process based on degenerative cell changes, affect tissues/ organs, deteriorate over time, normal bodily wear/ lifestyle”

– Wikipedia –

1. Degenerative Disease

Loss of normal histologic structures, **WITHOUT** significant infiltration inflammatory cells or proliferation abnormal cells

Ex: Osteoarthritis, atherosclerosis

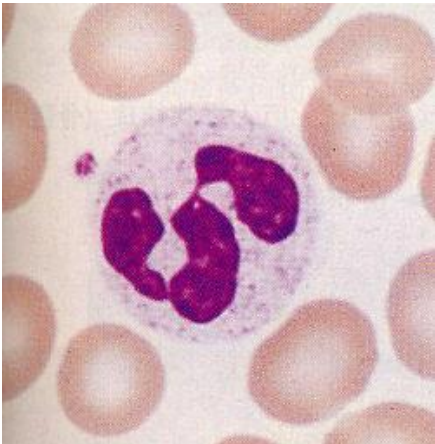
Assoc. with aging, hormonal involution, atrophy or hypoplasia

2. Inflammation

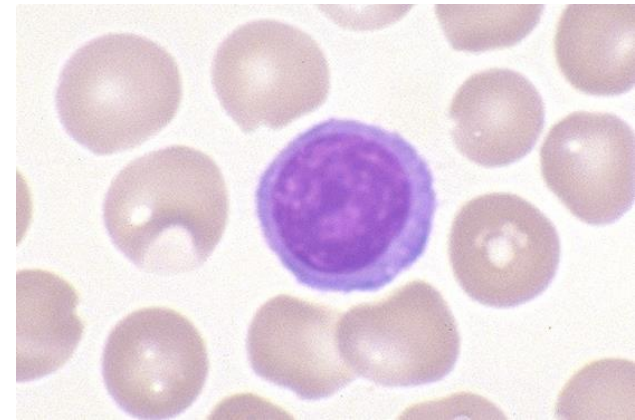
Cardinal Signs:

**Rubor – Calor – Dolor – Tumor –
Functiolaesa**

**Acute
Poly-morphnuclear
(PMN)**



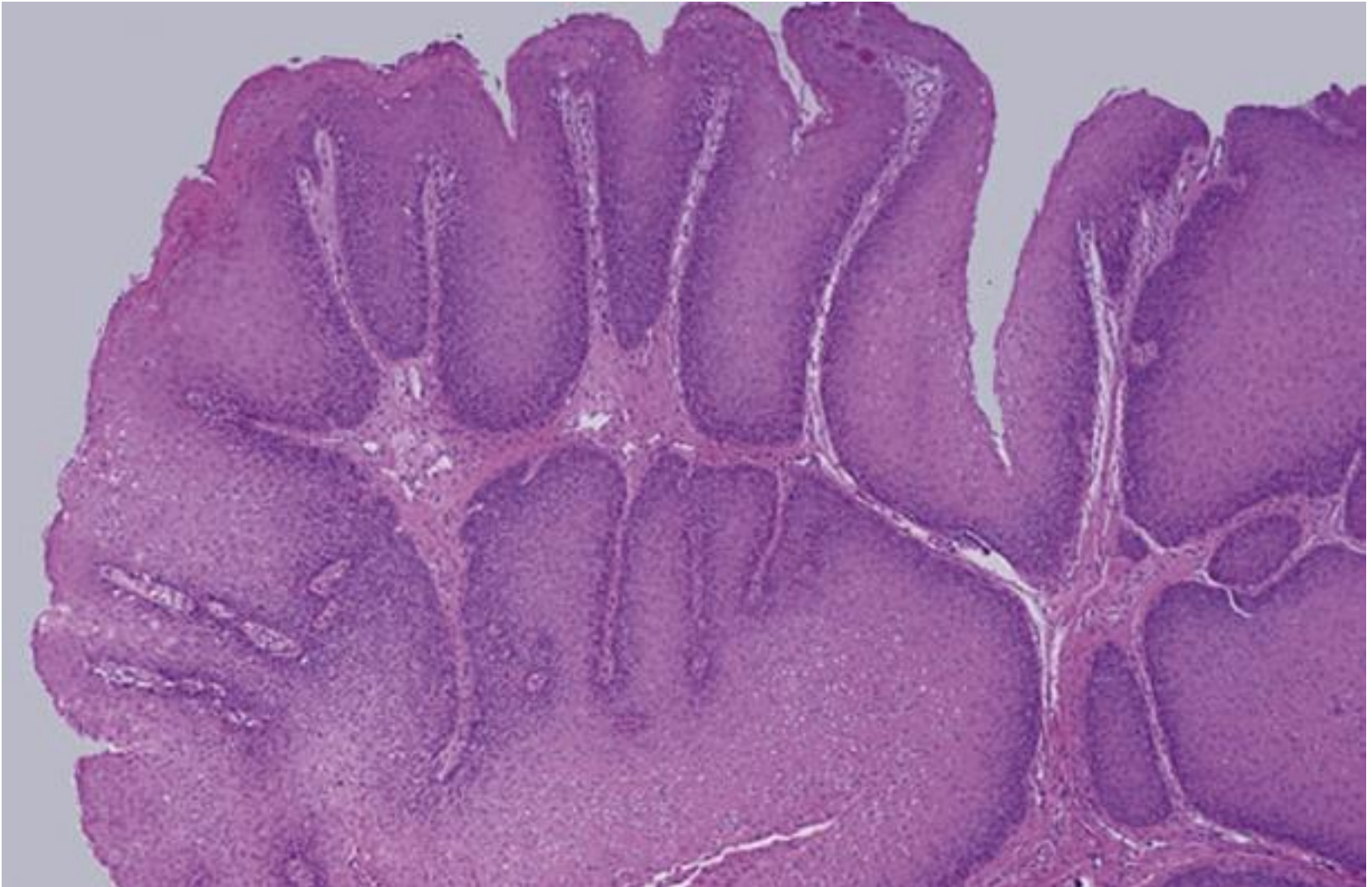
**Chronic
Mono-nuclear
(MN)**



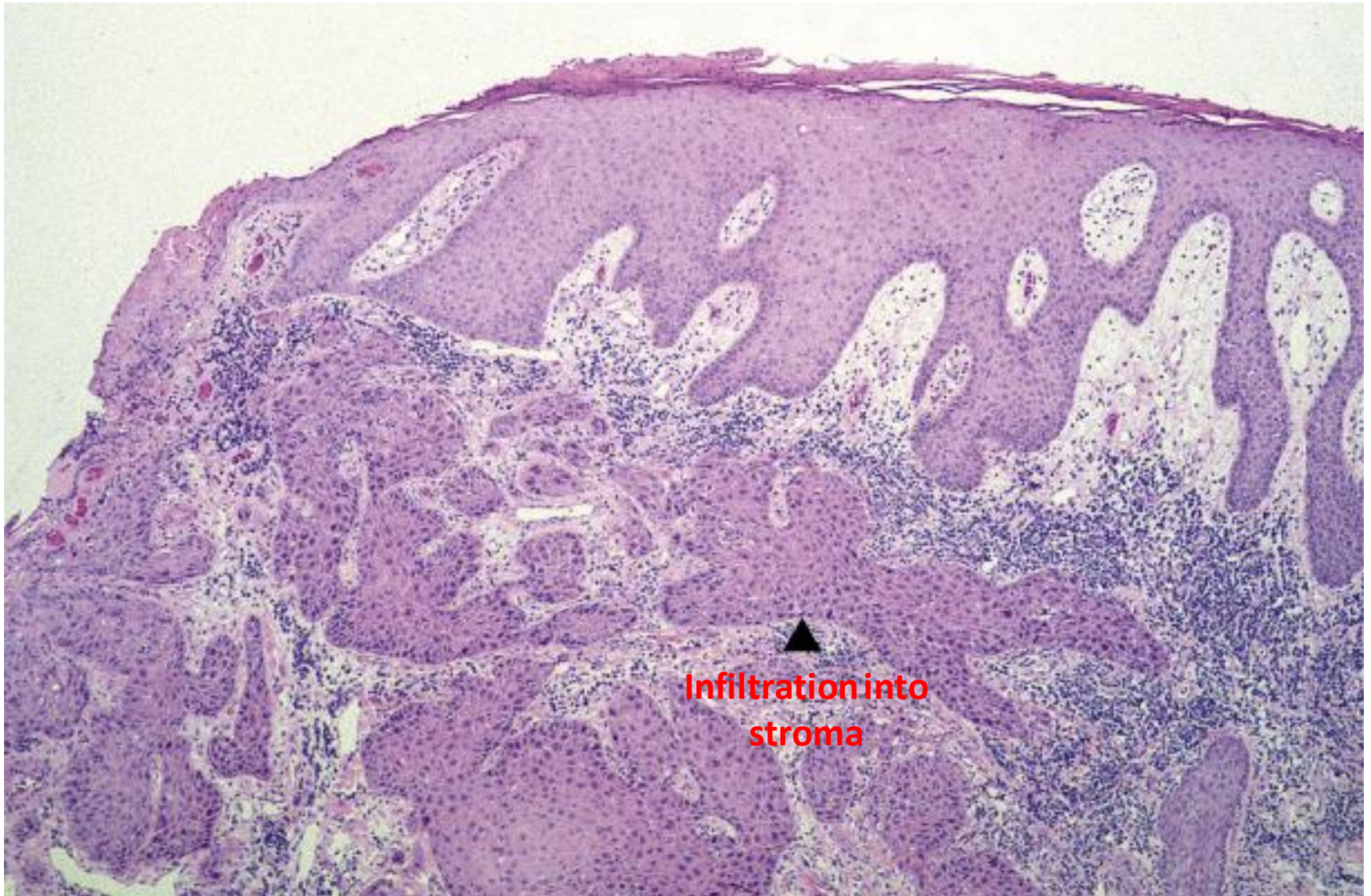
3. Neoplasm

- **Benign (No Infiltration into normal tissue)**
- **Malignant (Infiltration into normal tissue)**

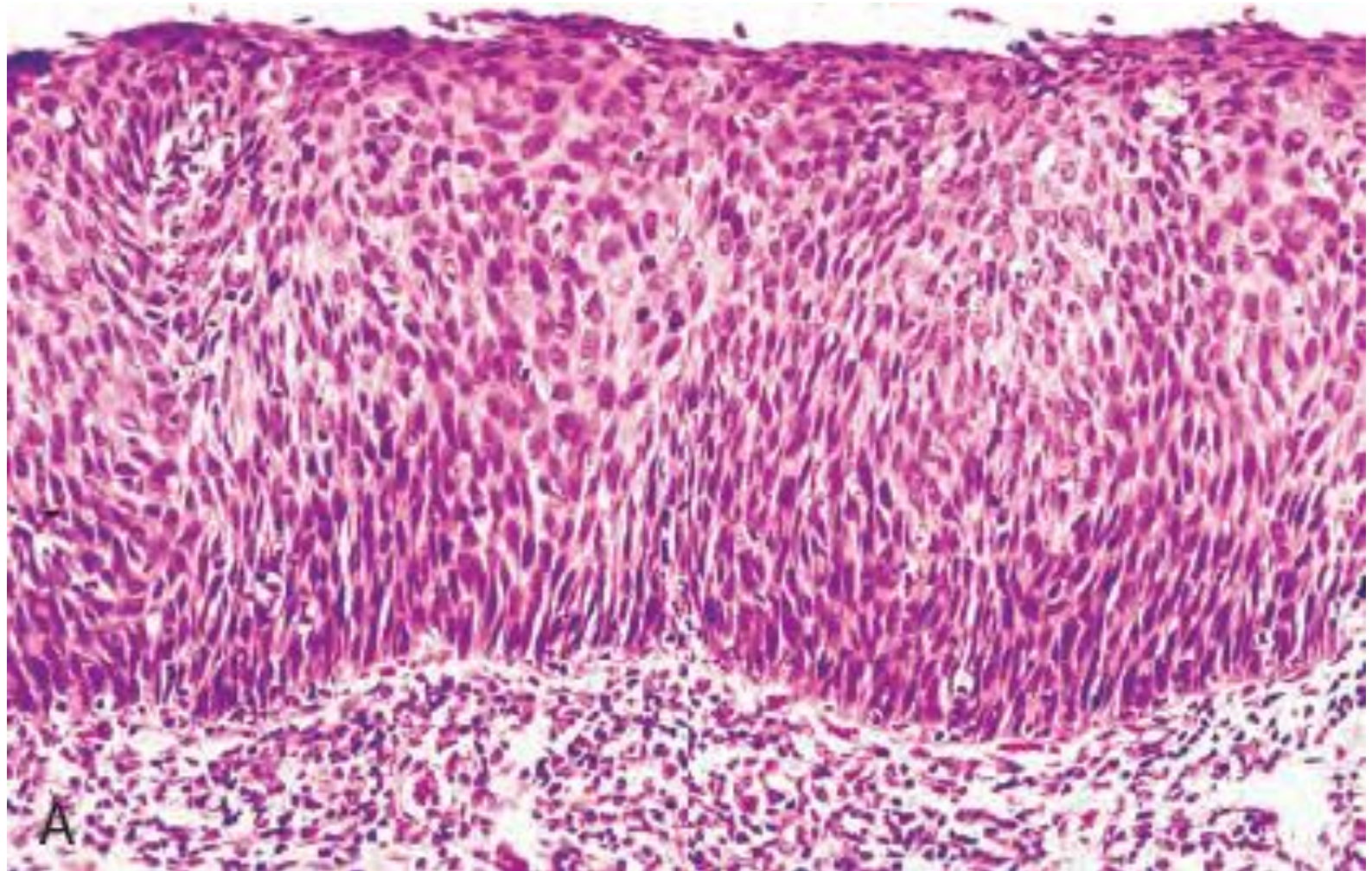
Benign Papilloma squamosa, nasal cavity



Malignant Squamous cell carcinoma, oral cavity



Pre-Malignant Servix, Squamous intraepithelial lesion



Conclusion..

NORMAL Histology

- Loss of normal cells → ?
- Infiltration by inflammatory cells (poly or mono) → ?
- Proliferation of abnormal cells → ?

General Pathology

BLOK 5

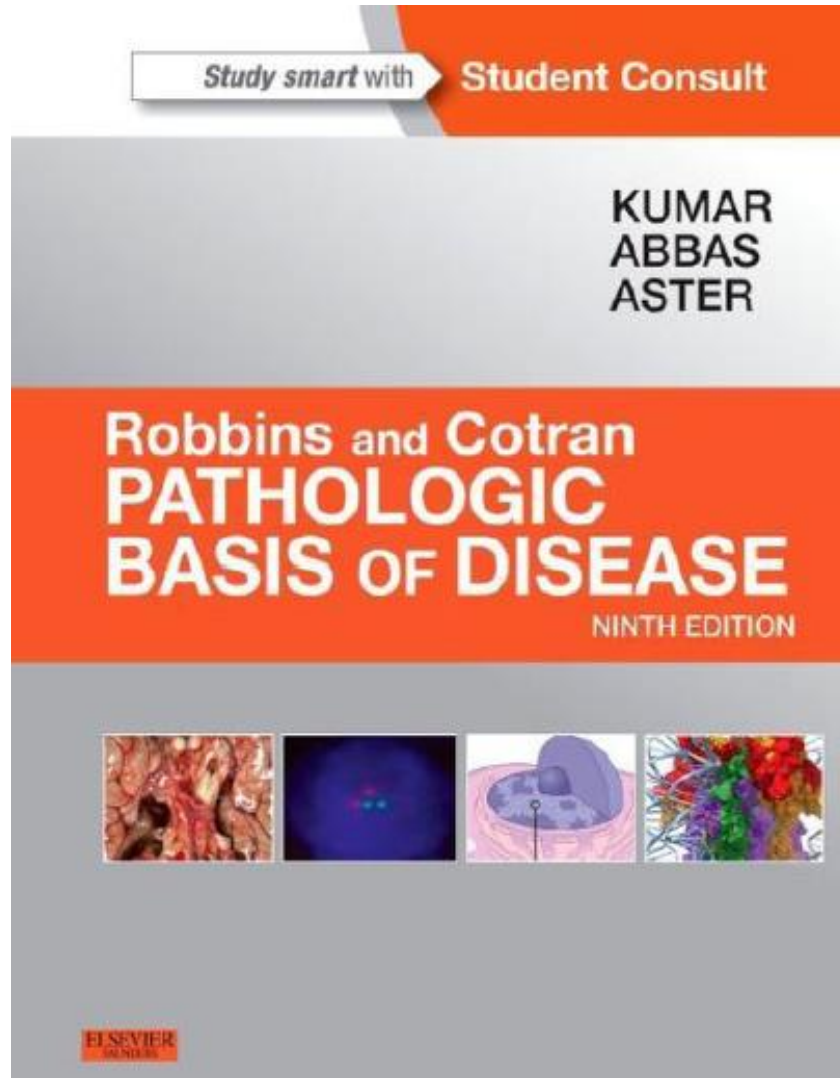
- **Cell Adaptations**
- **Inflammation: Acute, Chronic, etc,**
- **Regeneration/Healing**
- **Hemodynamic Disorders**
- **Genetic Diseases**
- **Immune system**
- **Neoplasms**
- **Infectious Diseases**
- ~~**Environmental/Nutritional Diseases**~~
- ~~**Childhood (Pediatric) Diseases**~~

Systemic Pathology

Blok 7, 8, 9.....

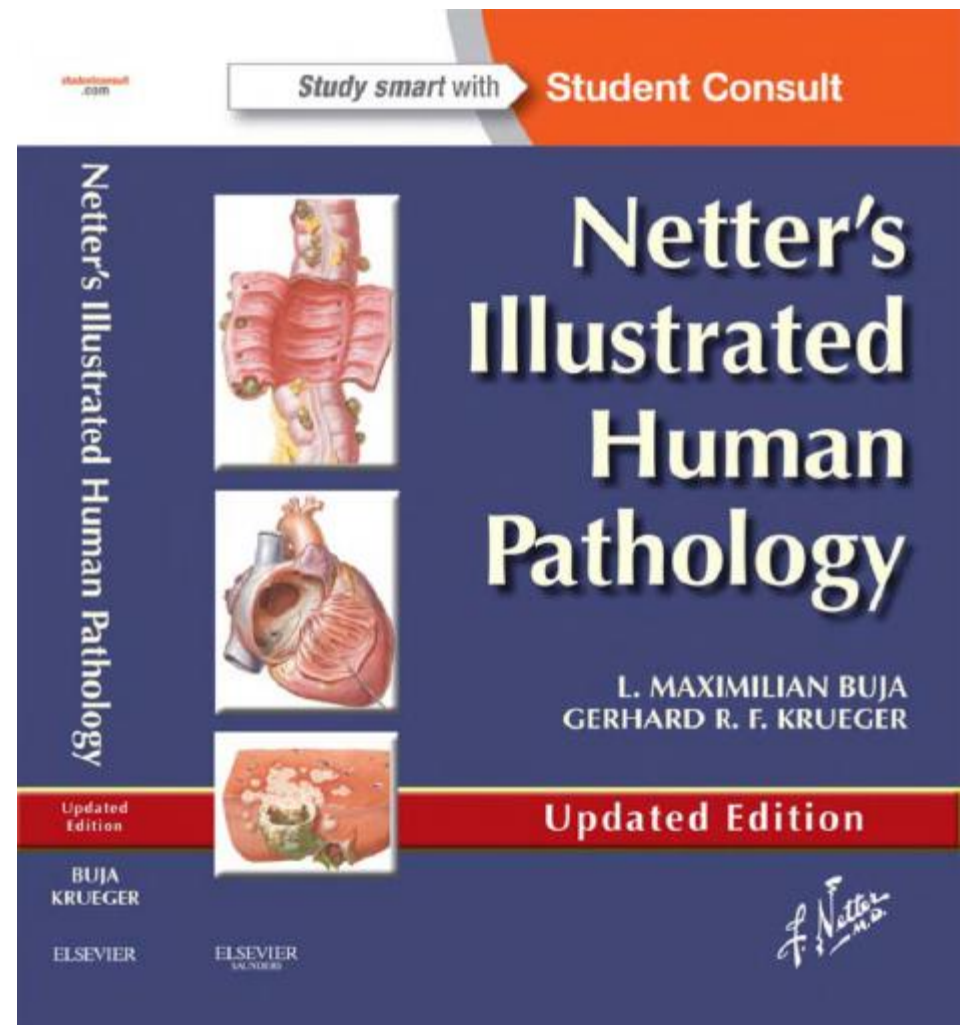
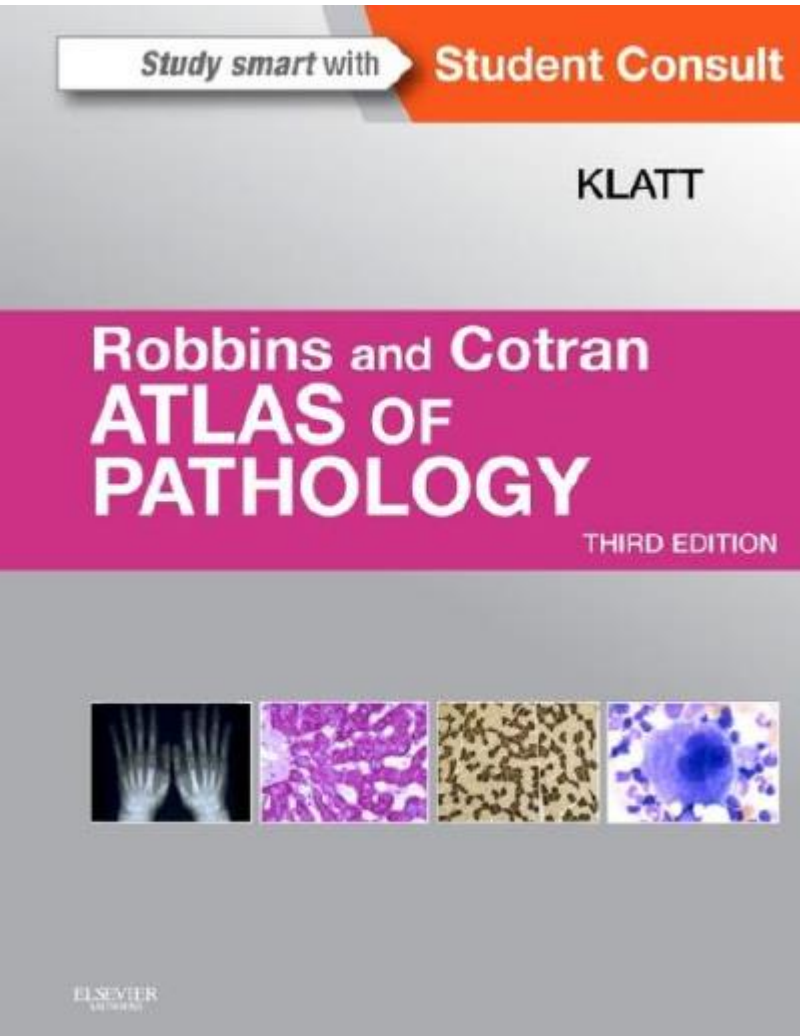
- Blood Vessels & Heart
- RBC & WBC
- Lung
- ENT
- GI – Liver - Pancreas
- Kidney
- Lower UT/Male
- **Female**
- **Breast**
- Endocrine
- Skin
- Orthopedic - Nerve/Muscle
- CNS
- Eye

The “Bible” of Pathology



- 9th Ed
- 29 chapters
- 1391 pages

Others..



Medical Specialty

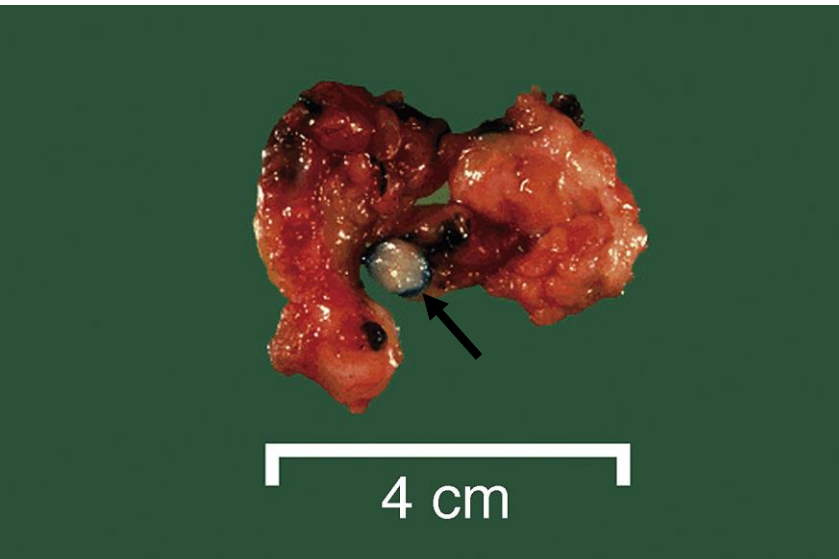
- **Anatomic Pathology → SpPA**
- **Clinical Pathology → SpPK**
- **Forensic Pathology → SpF**

Anatomic Pathology

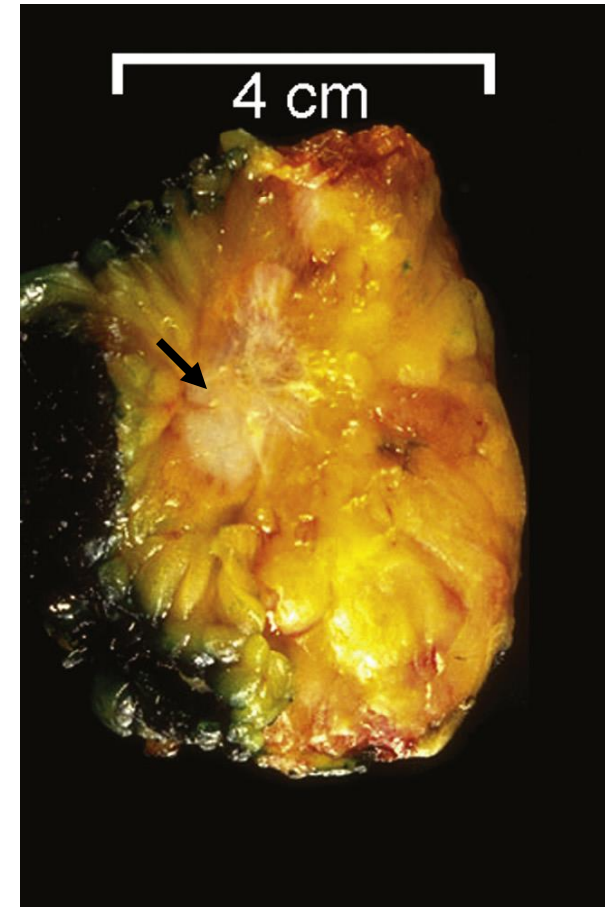
- **“Surgical” Pathology**
- **Cytology**
- **Clinic Autopsy**

“Surgical” Pathology

- Frozen section in operating theatre
- Grossing – macroscopic



VS

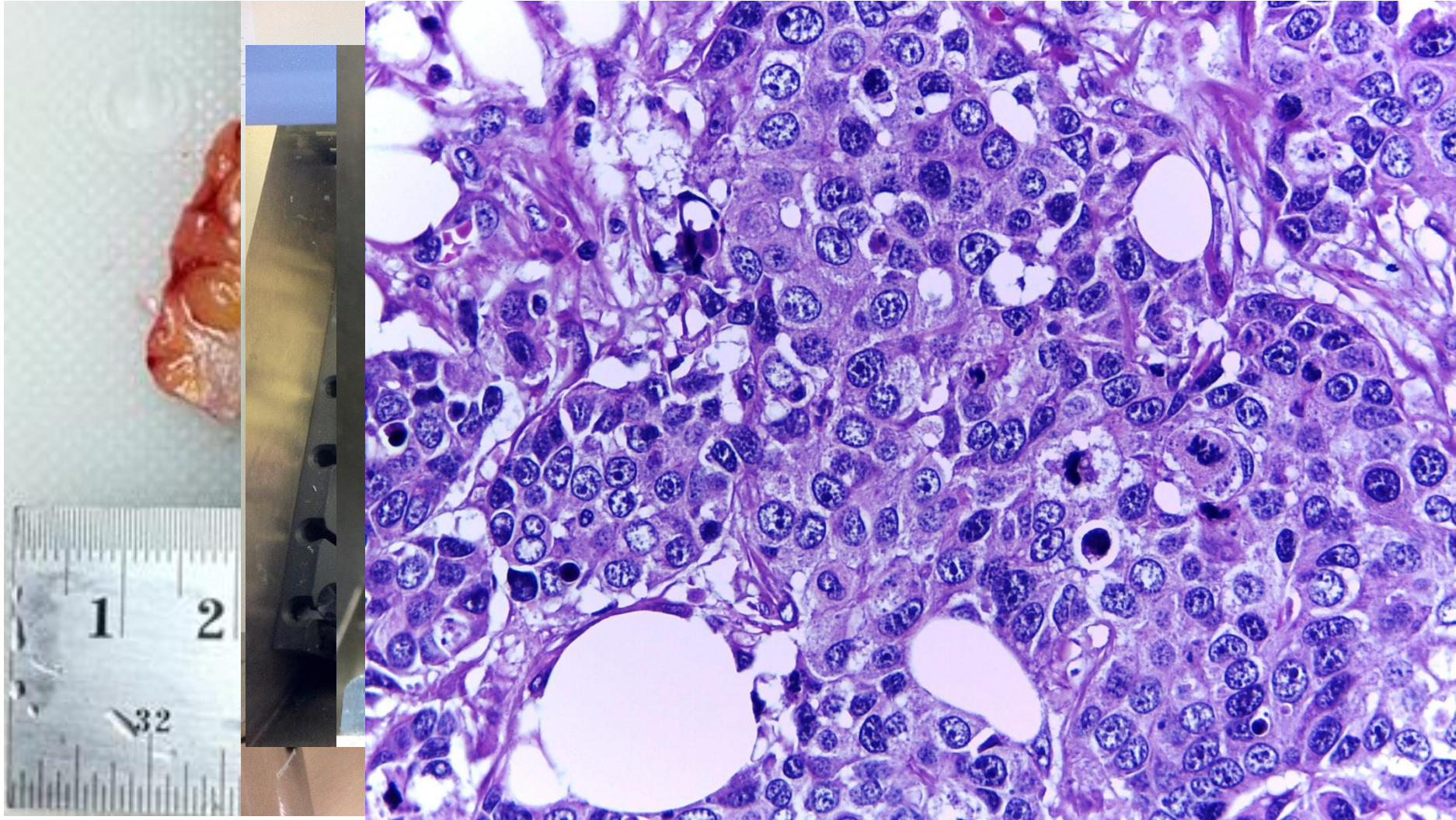


“Surgical” Pathology

Frozen Section

- Rapid Diagnosis during operation
- Defines what next for surgeon
- Mastectomy or Lumpectomy
- Lobectomy or total tiroidectomy

Frozen Section



♀ 55 yrs, Breast lump, ill defined border → benign or malignant?

Cytology

- Eksfoliasi: papsmear
- *Brushing/ sikatan (abrasive cytology)*
- **Biopsi Aspirasi Jarum Halus (*Fine Needle Aspiration Biopsy*): BAJAH/ FNAB**
- Intraoperasi

Biopsi Aspirasi Jarum Halus (BAJAH/ FNAB)

- Teknik aspirasi dan persiapan yang cermat.
- Organ-organ superfisial (palpasi atau panduan radiologis).
- Non-invasif, relatif mudah dilakukan, hasil cepat dan tidak mahal (*cost-effective*).

Biopsi Aspirasi Jarum Halus (BAJAH)/ Fine Needle Aspiration Biopsy (FNAB)

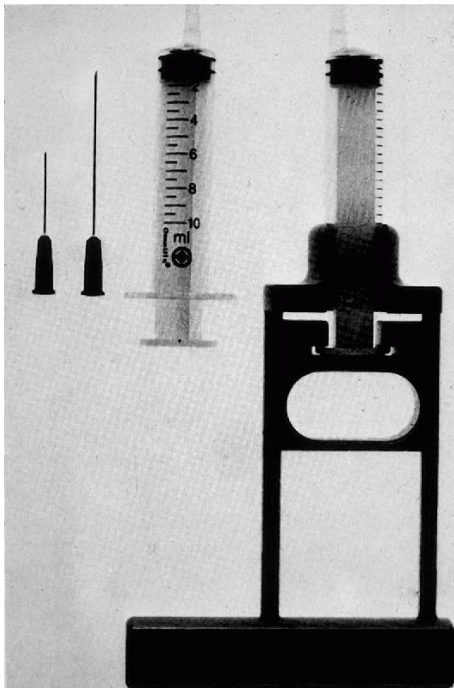


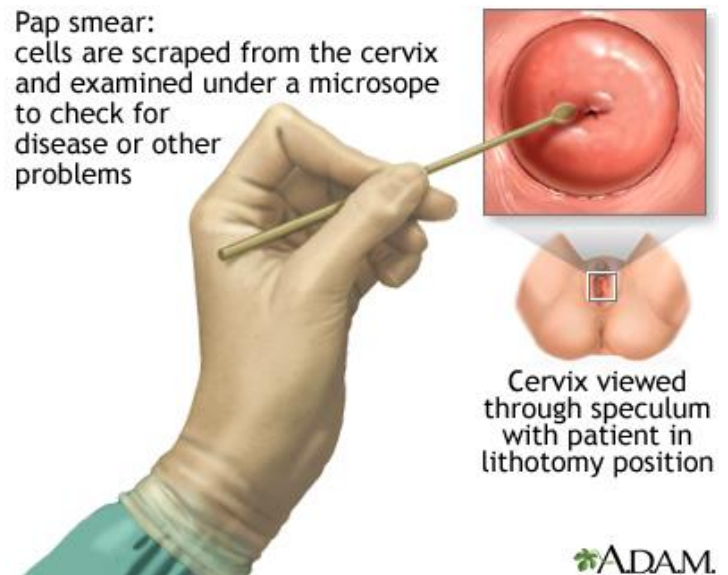
Fig. 2.1 FNB with aspiration (thyroid) Syringe and needle mounted in a pistol grip is operated by one hand, leaving the other free to feel and fix the target. Note the thumb supporting the syringe.

Intraoperasi

- Pemeriksaan sitologi potong beku (VC/ frozen section).
- *Rapid diagnosis, easier, faster and cheaper.*
- Panduan bagi ahli bedah apakah sudah mendapatkan lesi/ tumor, kelenjar getah bening *sentinel* pada ca mammae.
- Panduan untuk tatalaksana/ terapi selanjutnya.

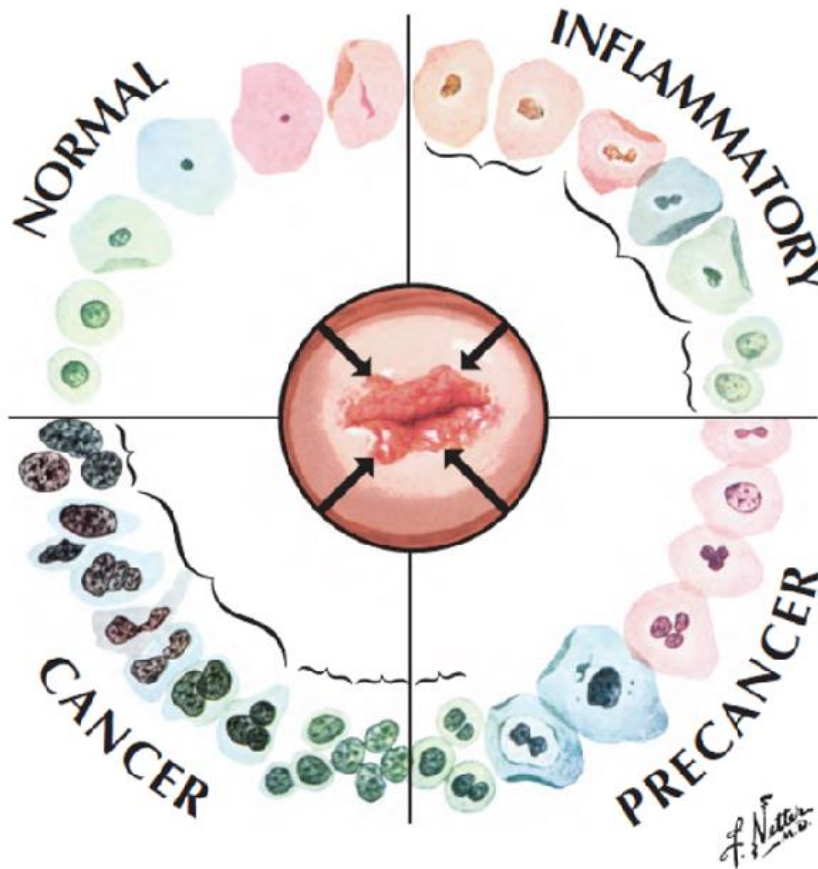
Pap Smear

- Program penapisan/ skrining skala besar.
- Efektif untuk pencegahan dan diagnosis kanker serviks.
- Konvensional – liquid based
- HPV *genotyping*

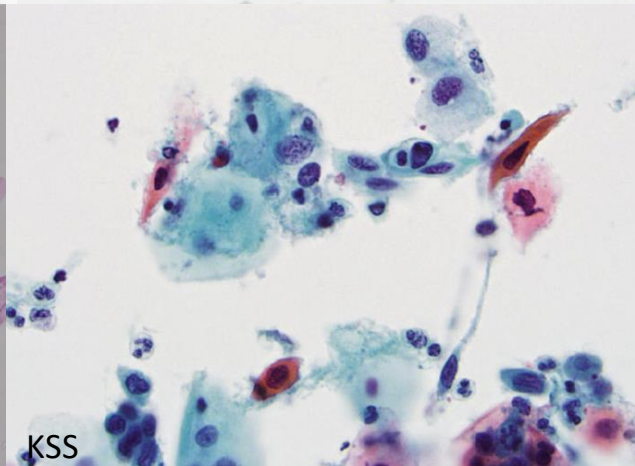
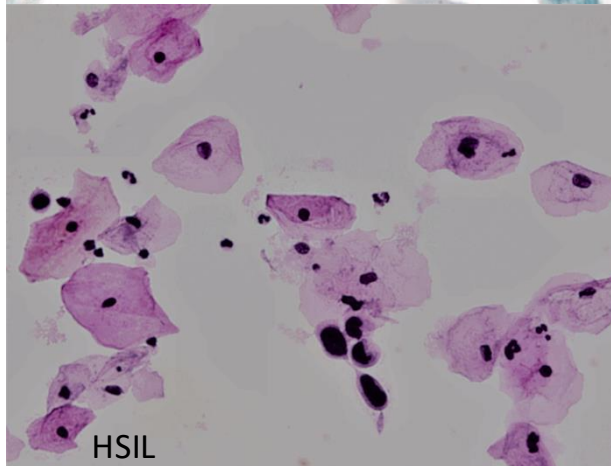
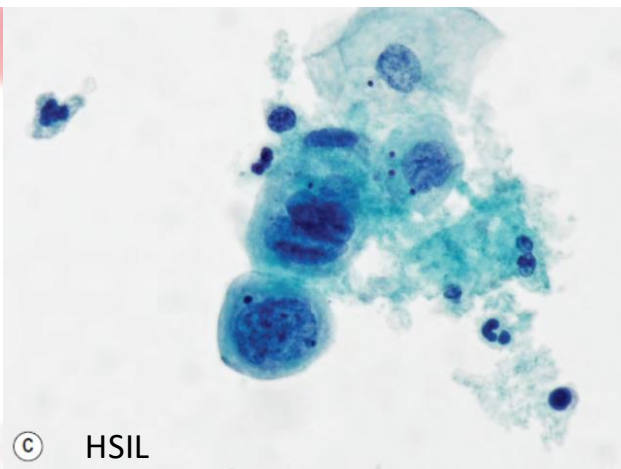
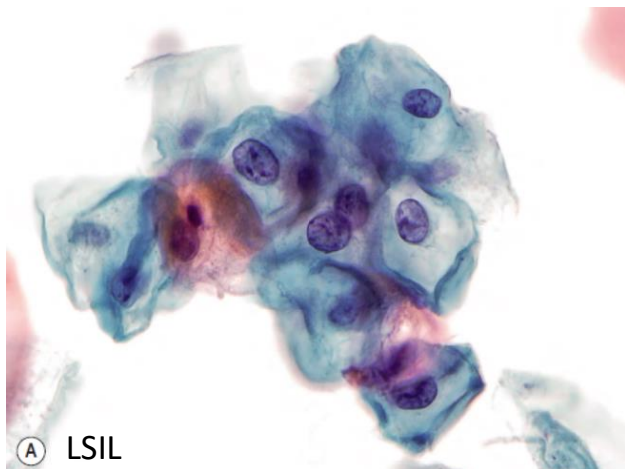


Cervical Cell Pathology in Squamous Tissue

Grades and cell types



- HPV (*Human Papilloma Virus*) 16, 18 menginfeksi sel epitel imatur.
- Lesi prakanker (**Cervical Intraepithelial Neoplasia**) :
 - CIN 1/ LSIL (**L**ow **G**rade **S**quamous **I**ntraepithelial **L**esion)
 - CIN 2 } HSIL (**H**igh **G**rade)
 - CIN 3 }
- Kanker → **K**arsinoma **S**el **S**kuamosa (KSS)



Clinic Autopsy

- Different with Forensic Autopsy
- Clinic autopsy:
medical cause of death (COD)
- Forensic Autopsy
Criminal, investigation cause of death
(COD)

Pathologist

- **Geeks**
- **Nerds**
- **Dorks**

Reasons to go into Pathology

- **Scientifically stimulation**

Basic science ----- Clinical Medicine

- **Broad range of skills and knowledge**
- **Connected to all parts of medicine**
- **Good hours, not much demanding call**
- **Ability to work well into 70s+**

Eye – Brain – Reports

Basic Pathology

6 weeks (April 19th – May 15th)

- 5 weeks:
 - Lectures, laboratory/ slide review
 - Hybrid: online and offline
 - Discussion – Presentation
- 1 weeks → **Examination**
Remedial...!!!

1

MINGGU - 1										
BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN										
WAKTU	SENIN 19/4		SELASA 20/4		RABU 21/4		KAMIS 22/4		JUMAT 23/4	
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10	T	KP PK I	KP PK 3	KP PK 2	BM	DKM	T	DKM	BM	PR <u>KKes</u>
09.30 – 11.10	KP PK I	T	KP PK 2	KP PK 3	DKM	PRAKT	DKM	T	PR <u>KKes</u>	BM
11.10 – 12.10	DKM	DKM	KON P	KON P	BM		BM	BM	IBADAH	
12.10 – 13.00	ISTIRAHAT/ MAKAN SIANG									
13.00 – 14.40	KP PA - 1	KP K Kes	KP PA 2	BM	PRAK	BM	KP PA 3	KP PK 4	DKM	KP PA 4
14.40 – 16.20	KP K Kes	KP PA - 1	BM	KP PA 2		BM	KP PK 4	KP PA 3	KP PA 4	DKM

Kuliah Pakar Patologi Anatomi:

1. Pendahuluan Patologi Anatomi: FAJ
2. Adaptasi Sel 1: MLG
3. Adaptasi Sel 2: MLG
4. Hemodinamik 1: MLG

Kuliah Pakar Patologi Klinik:

1. Kuliah Pengantar Patologi klinik, sampling dan blood collection
dr. Danny Luhulima SpPK
2. Hematopoiesis, dan leukosit: dr Erida Manalu SpPK
3. Morfologi Eritrosit (MCV, MCH, MCHC, Ht, LED, RDW, hipokromik, normokromik, makrositik, mikrositik): dr. Eirene SpPK
4. Morfologi Trombosit dan Gambaran darah tepi: dr. Danny Luhulima SpPK

PRAKTIKUM ONLINE

2

MINGGU - 2										
BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN										
WAKTU	SENIN 26/4		SELASA 27/4		RABU 28/4		KAMIS 29/4		JUMAT 30/4	
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10	T	KP PK 5	KP PK – 7	KP PK 6	BM	DKM	T	DKM	BM	PR <u>KKes</u>
09.30 – 11.10	KP PK 5	T	KP PK 6	KP PK 7	DKM	PRAKT	DKM	T	PR <u>KKes</u>	BM
11.10 – 12.10	DKM	DKM	KON P	KON P	BM		BM	BM	IBADAH	
12.10 – 13.00	ISTIRAHAT/ MAKAN SIANG									
13.00 – 14.40	KP PA – 5	KP K Kes	BM	KP PA 6	PRAK	BM	KP PA 7	KP PK 8	DKM	KP PA 8
14.40 – 16.20	KP K Kes	KP PA 5	KP PA 6	BM		BM	KP PK 8	KP PA 7	KP PA 8	DKM

Kuliah Pakar Patologi Anatomi:

- Hemodinamik 2: FAJ
- Radang 1: BRH
- Radang 2: FAJ
- Regenerasi 1: MLG

Kuliah Pakar Patologi Klinik:

- Hemostasis (dr. Danny Luhulima SpPK)
- Anemia (dr. Danny Luhulima SpPK)
- Hematoanalyzer, Bone Marrow Aspiration dan pemeriksaan manual sel darah dr. Eirene T SpPK
- Golongan darah Transfusi dan hemoglobinopathy: dr Erida Manalu SpPK

3

MINGGU - 3										
BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN										
WAKTU	SENIN 3/5		SELASA 4/5		RABU 5/5		KAMIS 6/5		JUMAT 7/5	
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10	T	KP PK 9	KP PK 11	KP PK 10	BM	DKM	T	DKM	BM	PR <u>KKes</u>
09.30 – 11.10	KP PK 9	T	KP PK 10	KP PA 11	DKM	PRAKT	DKM	T	PR <u>KKes</u>	BM
11.10 – 12.10	DKM	DKM	KON P	KON P	BM		BM		IBADAH	
12.10 – 13.00	ISTIRAHAT/ MAKAN SIANG									
13.00 – 14.40	KP PA 9	KP K Kes	BM	KP PA 10	PRAKT	BM	KP PA 11	KP PK 12	DKM	KP PA 12
14.40 – 16.20	KP K Kes	KP PA 9	KP PA 10	BM		BM	KP PK 12	KP PA 11	KP PA 12	DKM

Kuliah Pakar Patologi Anatomi:

- Regenerasi 2: MLG
- Infeksi: MDN
- Patologi molekular/ genetik: NUR
- Onkogenesis: MLG

Kuliah Pakar Patologi Klinik:

- Ginjal dan saluran Kemih (dr. Erida Manalu SpPK)
 - Urinalisis (dr Eirene T SpPK)
 - Analisis Cairan tubuh (pleura, otak, sendi, ascites) (dr. Danny Luhulima SpPK)
 - Lipid profile (Dr Eirene T SpPK)
- TUGAS:** Manfaat klinis : Ur, Cr, AST, ALT, Lipid profile, Elektrolit, Protein, Urinalisis dan Cairan tubuh (Tugas membuat paper dengan tulisan tangan)

4

MINGGU - 4										
BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN										
WAKTU	SENIN 10/5		SELASA 11/5		RABU 12/5		KAMIS 13/5		JUMAT 14/5	
	LIBUR HARI RAYA									
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10										
09.30 – 11.10										
11.10 – 12.10										
12.10 – 13.00										
13.00 – 14.40										
14.40 – 16.20										

5

MINGGU - 5 BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN

WAKTU	SENIN 17/5		SELASA 18/5		RABU 19/5		KAMIS 20/5		JUMAT 21/5	
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10	T	KP PK 13	KP PK 15	KP PK 14	BM	DKM	T	DKM	BM	PR K <u>Kes</u>
09.30 – 11.10	KP PK 13	T	KP PK 14	KP PK 15	DKM	PRAKT	DKM	T	PR K <u>Kes</u>	BM
11.10 – 12.10	DKM	DKM	KON P	KON P	BM		BM		IBADAH	
12.10 – 13.00	ISTIRAHAT/ MAKAN SIANG									
13.00 – 14.40	KP PA 13	KP K Kes	BM	KP PA 14	PRAK	BM	KP PK 16	KP PA 15	DKM	KP PA 16
14.40 – 16.20	KP K Kes	KP PA 13	KP PA 14	BM		BM	KP PA 15	KP PK 16	KP PA 16	DKM

Kuliah Pakar Patologi Anatomi:

Kuliah Pakar Patologi Klinik

13. Neoplasma: FAJ
14. Payudara: FAJ
15. Serviks: MLG
16. Sistem imun: FAJ

13. Faal Pemeriksaan Hati (AST, ALT, Gamma GT, Amilase, Lipase, Albumin-Globulin) (dr. Erida Manalu SpPK)
14. Elektrolit dan Protein: (dr. Eirene T SpPK)
15. Imunologi Dasar (dr. Danny Luhulima SpPK)
16. Tiroid: (dr. Erida Manalu SpPK)

PRAKTIKUM TATAPMUKA/ OFFLINE

6

MINGGU - 6 BIOMEDIK (PK + PA) DAN KOMUNIKASI KESEHATAN

WAKTU	SENIN 24/5		SELASA 25/5		RABU 26/5		KAMIS 27/5		JUMAT 28/5	
	A	B	A	B	A	B	A	B	A	B
07.30 – 9.10	T	KP PK 17	KP PK 19	KP PK 18	LIBUR	WAISAK	T	DKM	BM	PR <u>KKes</u>
09.30 – 11.10	KP PK 17	T	KP PK 18	KP PK 19			DKM	T	PR <u>KKes</u>	BM
11.10 – 12.10	DKM	DKM	KON P	KON P			BM		IBADAH	
12.10 – 13.00	ISTIRAHAT/ MAKAN SIANG									
13.00 – 14.40	KP PA 17	KP K Kes	BM	KP PK 18			KP PK 20	KP PA 19	DKM	KP PA 20
14.40 – 16.20	KP K Kes	KP PA 17	KP PA 18	BM			KP PA 19	KP PK 20	KP PA 20	DKM

Kuliah Pakar Patologi Anatomi:

17. Video pemrosesan jaringan PA: FAJ
18. Peranan PA dalam diagnosis dan terapi kanker
19. Kapita selekta/ Praktikum: Tim PA
20. Kapita selekta/ Praktikum: Tim PA

Kuliah Pakar Patologi Klinik:

17. Immunoassay (ELISA, ICT), PCR dan fotometer: dr. Erida Manalu SpPK
18. Karbohidrat, Glukosa, DM dan *metabolic syndrome* (dr. Eirene T SpPK)
19. Enzim Saluran Cerna dan analisis feces: (dr. Danny Luhulima SpPK)
20. Leukemia (dr. Eirene T SpPK)

PRAKTIKUM ONLINE

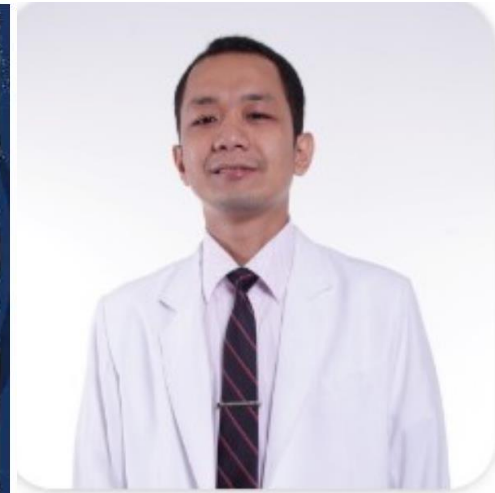
Remember!!!

There are No magic formulas for academic achievement.

The most important thing you can do is to spend some time each day in learning process.

Edward C. Klatt MD

Vinay Kumar MD



**TERIMA KASIH..HATUR
NUHUN..MAULIATE..**



Universitas Kristen Indonesia

Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 073/UKI.F5.D/HKP.3.5.6/2021
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

- MENIMBANG** : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendid di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk member Kuliah Pakar
- MENGINGAT** : 1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KB FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 ten pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

- MENETAPKAN** : 1. Penugasan dalam memberikan Kuliah Pakar :
- | | |
|--------------|------------------------------|
| Nama | dr. Fajar L. Gultom, Sp.PA |
| Departemen | Patologi Anatomi |
| Blok | 5 (Biomedik) |
| Judul Materi | Neoplasma |
| Semester | genap 2020/2021 |
| Kelas | A : 0,21 SKS
B : 0,21 SKS |
| SKS | 0,42 SKS |
2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputi ini akan diperbaiki sebagaimana mestinya

Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 15 April 2021
Dekan,


Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:

1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI

● RENDAH HATI ● BERBAGI DAN PEDULI ● PROFESIONAL ● BERTANGGUNG JAWAB ● DIS



**WORLD
CANCER
DAY 2016**



WORLD Cancer DAY.ORG

#WorldCancerDay
#WeCanI Can

WE CAN. I CAN.

**JOIN US
ON 4 FEB**

Neoplasma

Fajar L. Gultom
Departemen Patologi Anatomi
FKUKI
Mei 2021

Definisi

- Neo – baru
- Plasia – pertumbuhan
- Pertumbuhan baru – new growth
- Neoplasma – tumor
- Oncologi → oncos - logos



Neoplasia

Benign VS Malignant Tumor behavior Clinically

Neoplasia

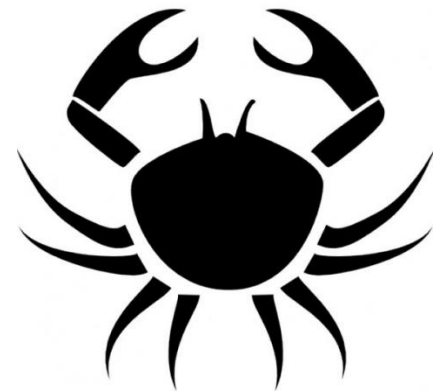
Benign

- Macros dan micros: *innocent*
- localized
- Excision → recurrence <<
- Survival >>

Neoplasia

Malignant

- *Cancers* – crab
- Invade and destroy adjacent structures
- Spread to distant sites → Metastasize → †



Benign Neoplasm

- Suffix.... **oma**
- Fibro---oma
- Lipo---oma
- Hemangi---oma
- Kondro---oma
- Mio---oma
- Glands → adenoma
- Papil---oma
- Cyst in ovary → cystadenoma

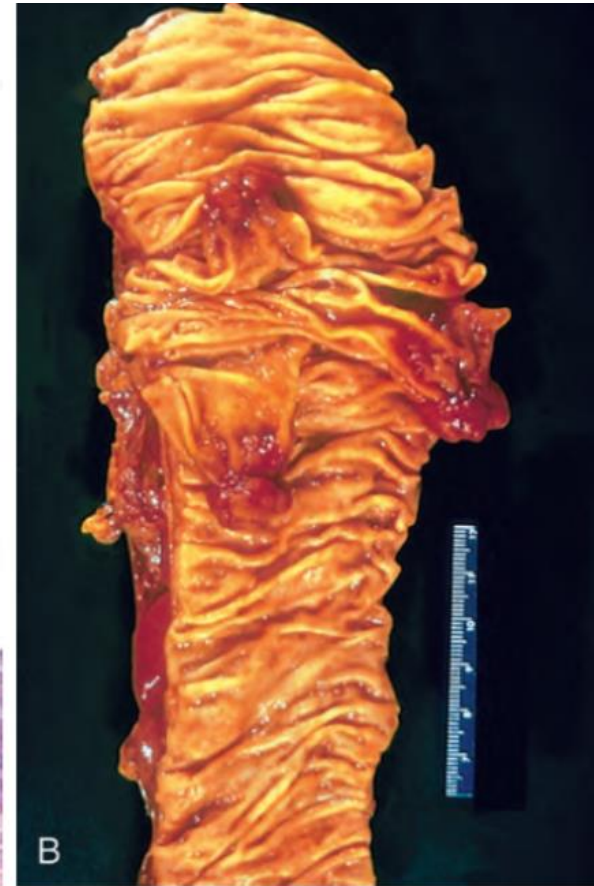
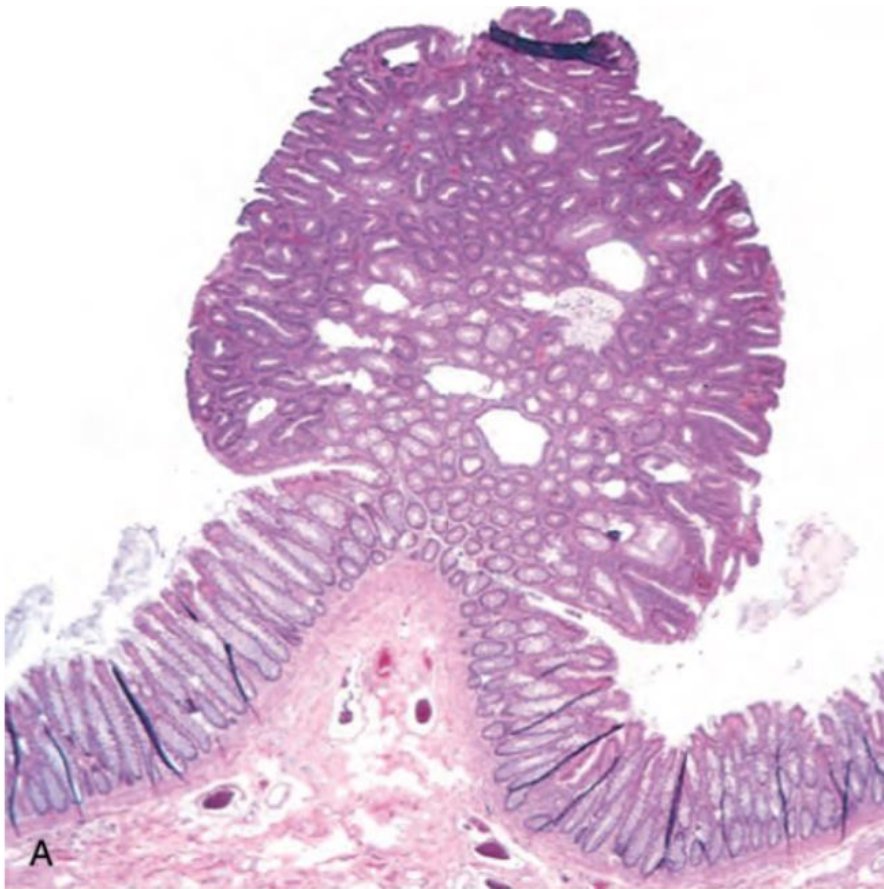
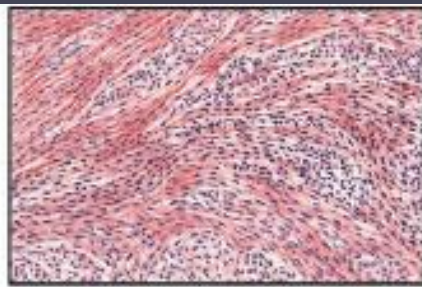
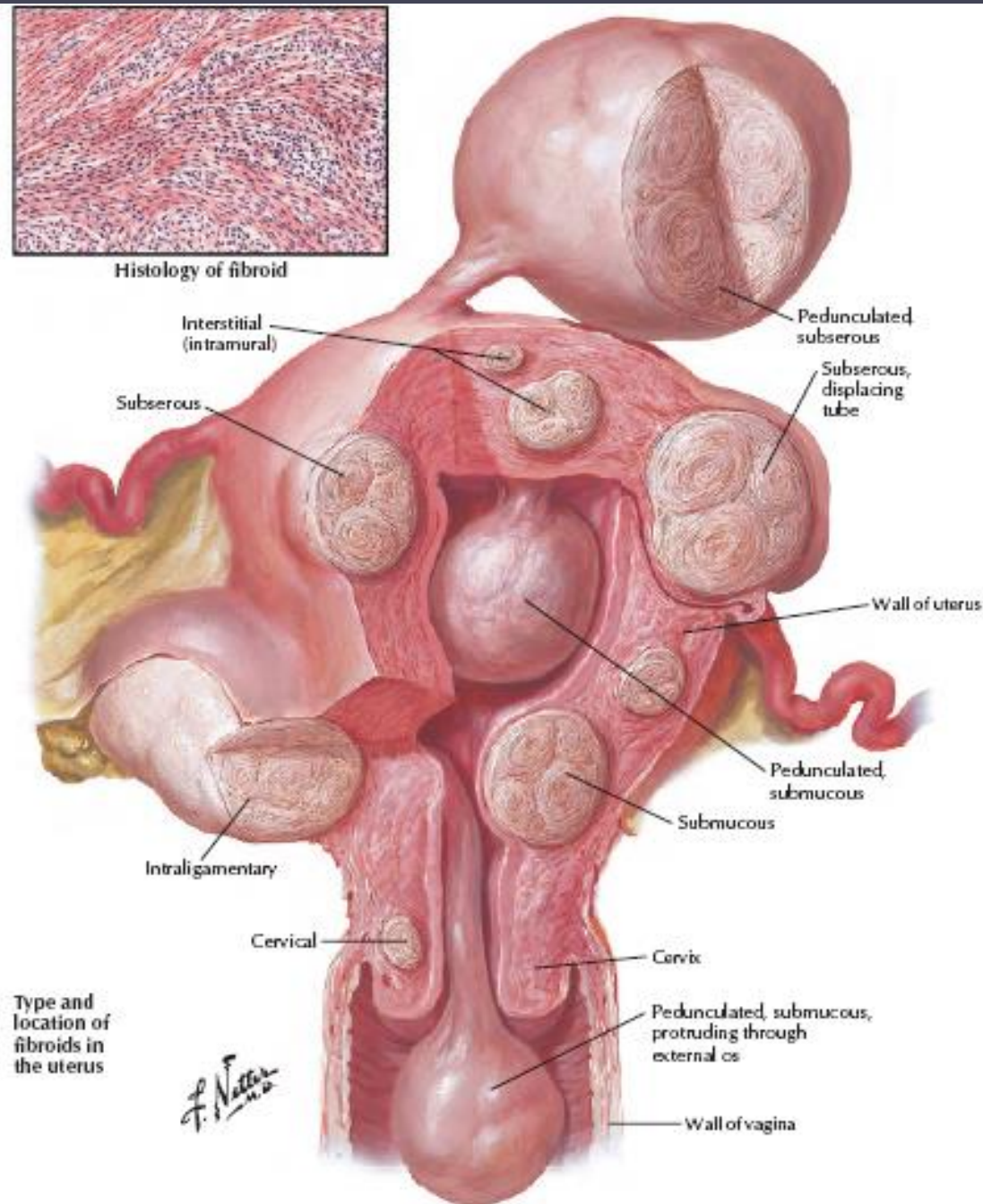


Figure 7-1 Colonic polyp. **A**, An adenomatous (glandular) polyp is projecting into the colonic lumen and is attached to the mucosa by a distinct stalk. **B**, Gross appearance of several colonic polyps.



Histology of fibroid



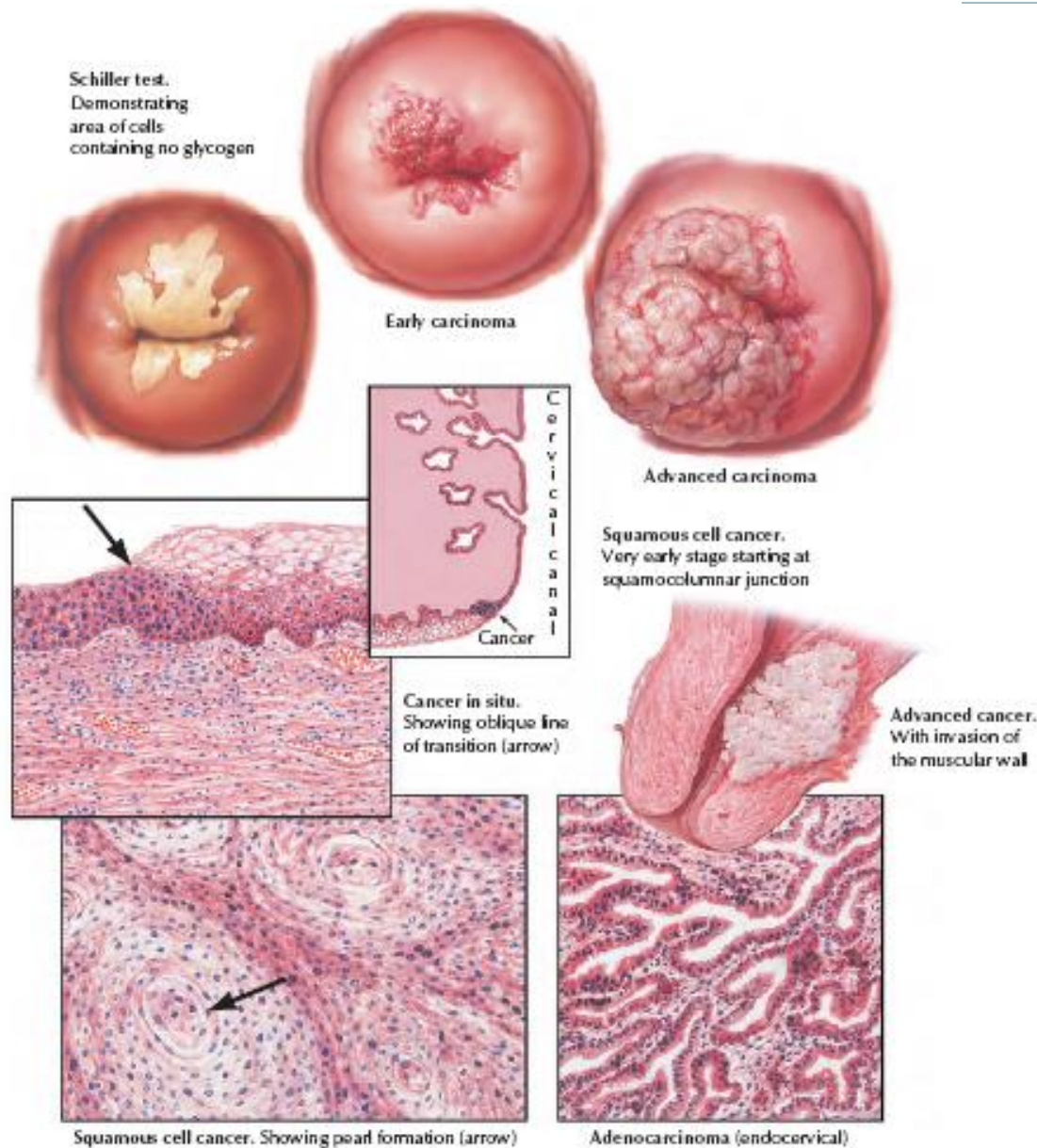
Type and location of fibroids in the uterus

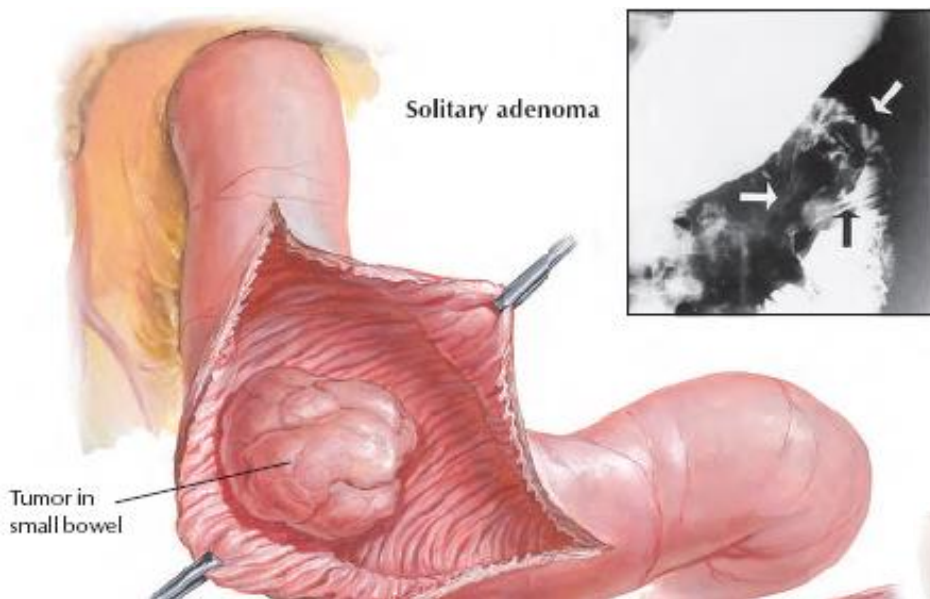
Malignant Neoplasm

- Carcinoma – epithelial (squamous, columnar, transitional)
 - Squamous cell carcinoma, adenocarcinoma
- Sarcoma – mesenchymal (connective tissue: cartilage, bone, vessels, adipose, fibrous tissue, nerve)
 - Osteosarcoma, chondrosarcoma, liposarcoma
- Hematolymphoid – leukemia, lymphoma

Cervical cancer:

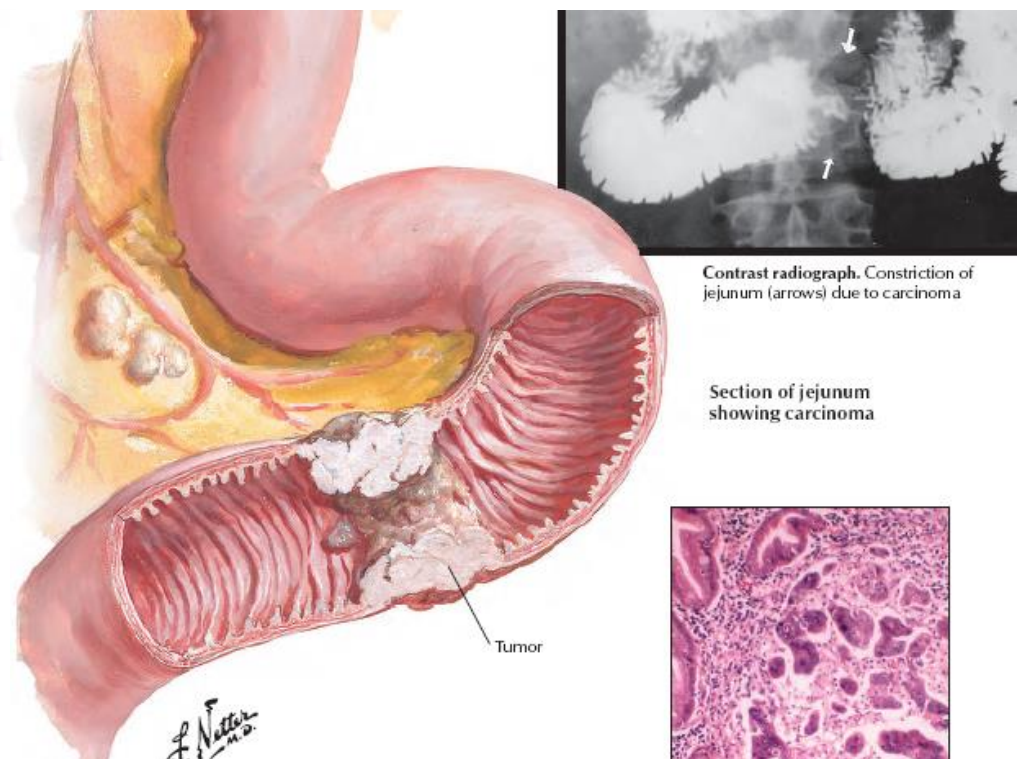
- Squamous cell carcinoma
- Adenocarcinoma





Benign tumor in small bowel

Malignant tumor in small bowel



Mixed Tumor

- Contain epithelial component and mesenchymal component
- Salivary gland: pleomorphic adenoma
- Ovary: teratoma

Teratoma Ovary

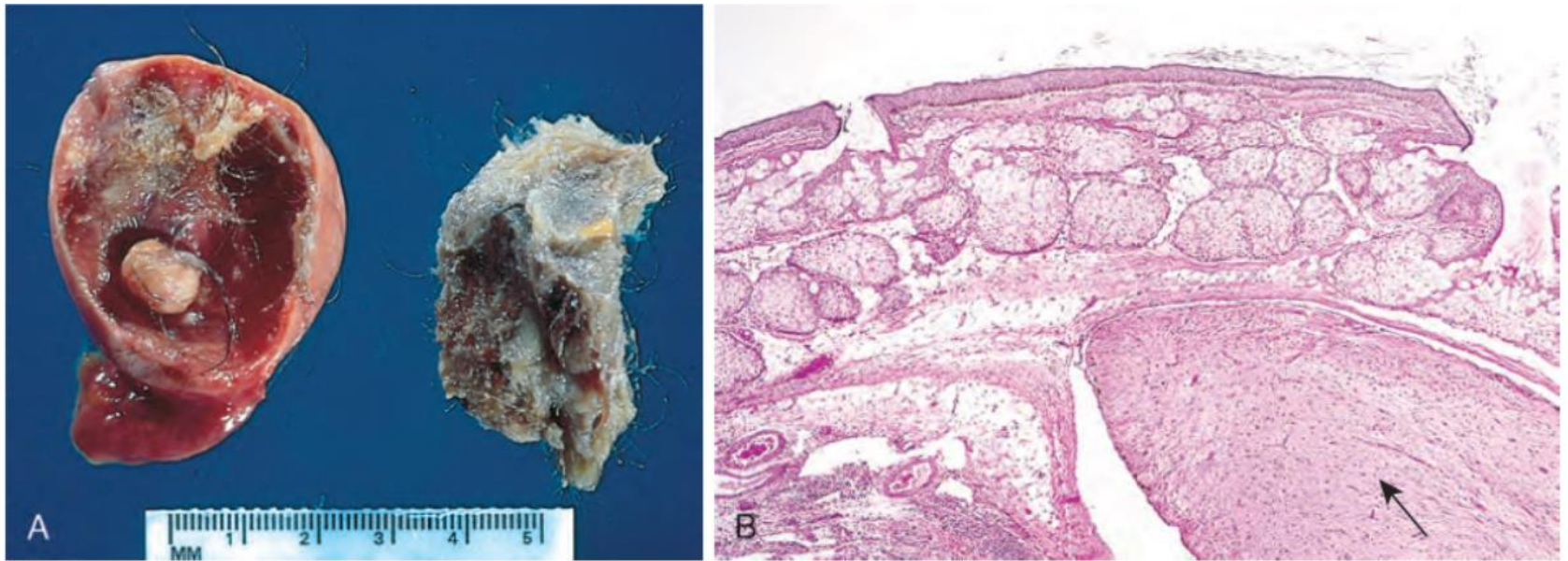


Figure 7-3 **A**, Gross appearance of an opened cystic teratoma of the ovary. Note the presence of hair, sebaceous material, and tooth. **B**, A microscopic view of a similar tumor shows skin, sebaceous glands, fat cells, and a tract of neural tissue (*arrow*).

Neoplasm

- Differentiation and anaplasia
- Rate of growth
- Local invasion
- Metastasize

Differentiation and Anaplasia

- Sel tumor – normal counterpart
- Jinak – mirip sel normal
Lipoma, kondroma, leiomioma
- Ganas – diferensiasi luas: well diff – poor diff
Karsinoma sel skuamosa serviks
Adenokarsinoma kolon

Differentiation and Anaplasia

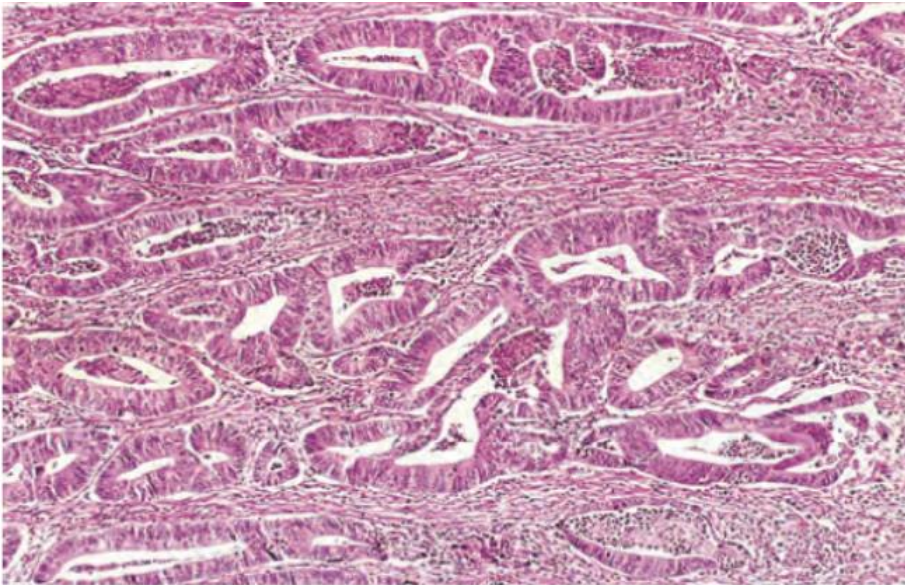


Figure 7-6 Malignant tumor (adenocarcinoma) of the colon. Note that compared with the well-formed and normal-looking glands characteristic of a benign tumor (Fig. 7-5), the cancerous glands are irregular in shape and size and do not resemble the normal colonic glands. This tumor is considered differentiated because gland formation is seen. The malignant glands have invaded the muscular layer of the colon. (Courtesy Dr. Trace Worrell, University of Texas Southwestern Medical School, Dallas, Texas.)

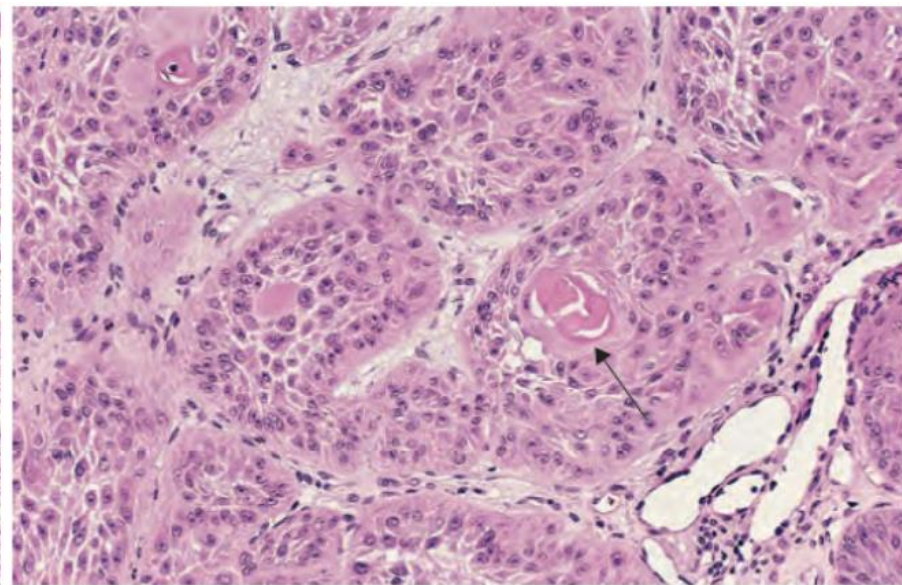


Figure 7-7 Well-differentiated squamous cell carcinoma of the skin. The tumor cells are strikingly similar to normal squamous epithelial cells, with intercellular bridges and nests of keratin pearls (*arrow*). (Courtesy Dr. Trace Worrell, University of Texas Southwestern Medical School, Dallas, Texas.)

Differentiation and Anaplasia

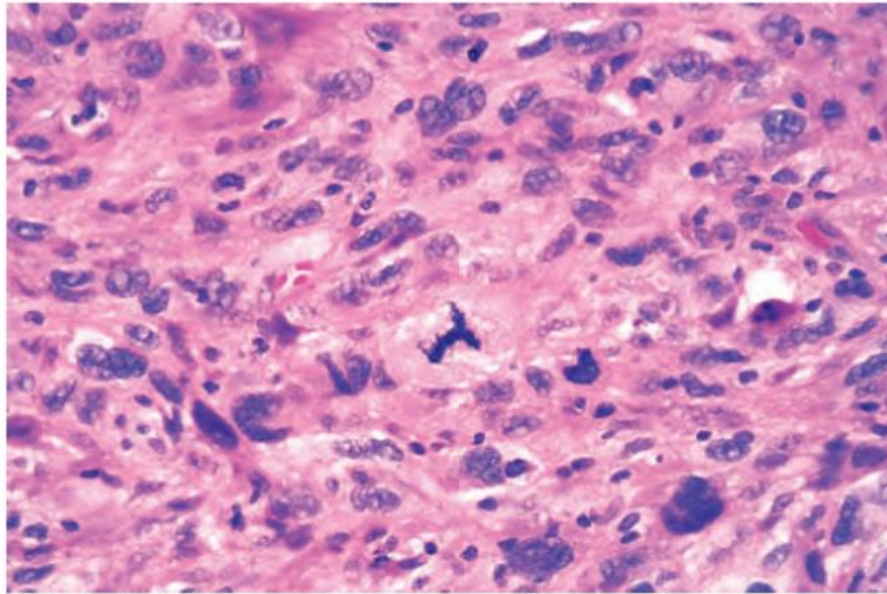


Figure 7-8 Anaplastic tumor showing cellular and nuclear variation in size and shape. The prominent cell in the center field has an abnormal tripolar spindle.

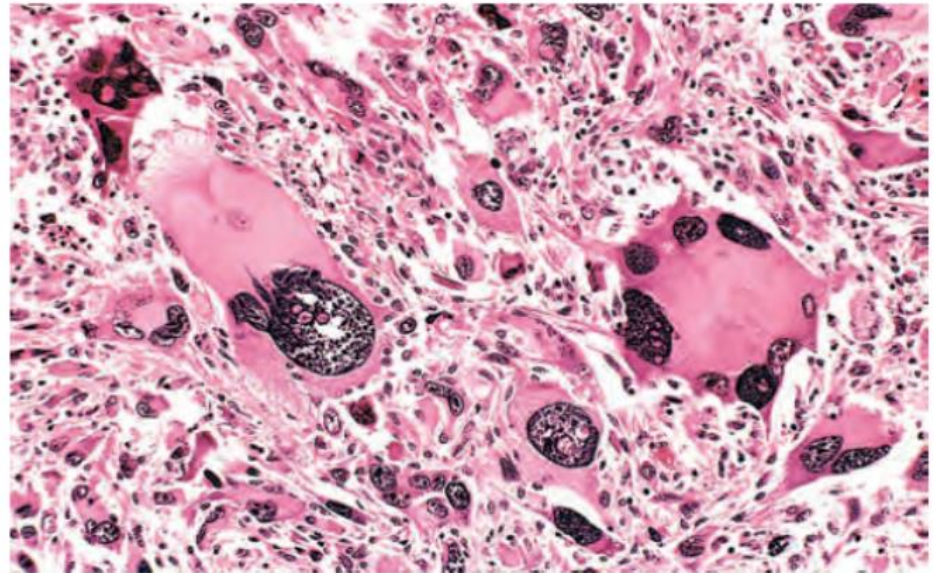


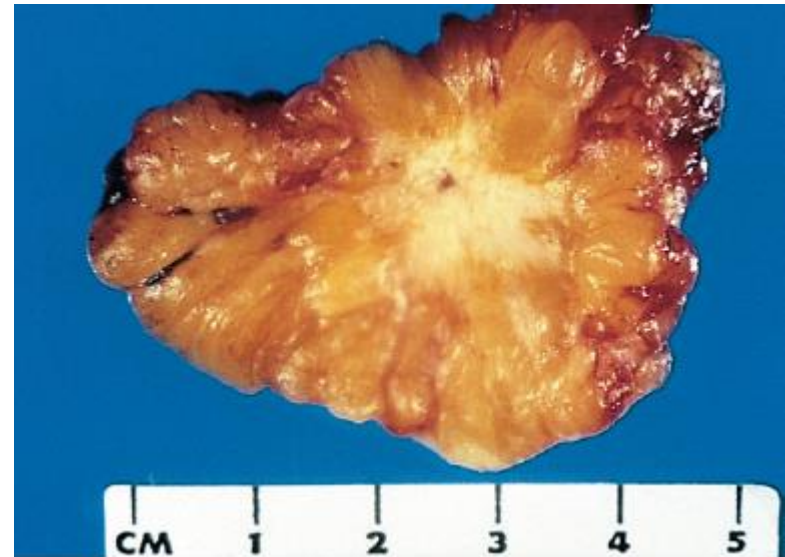
Figure 7-9 Pleomorphic tumor of the skeletal muscle (rhabdomyosarcoma). Note the marked cellular and nuclear pleomorphism, hyperchromatic nuclei, and tumor giant cells. (Courtesy Dr. Trace Worrell, University of Texas Southwestern Medical School, Dallas, Texas.)

Rate of Growth

- Jinak – lambat - aliran darah, hormon fibroadenoma mammae (FAM), leiomioma pada kehamilan
- Ganas – tergantung diferensiasinya

Local Invasion

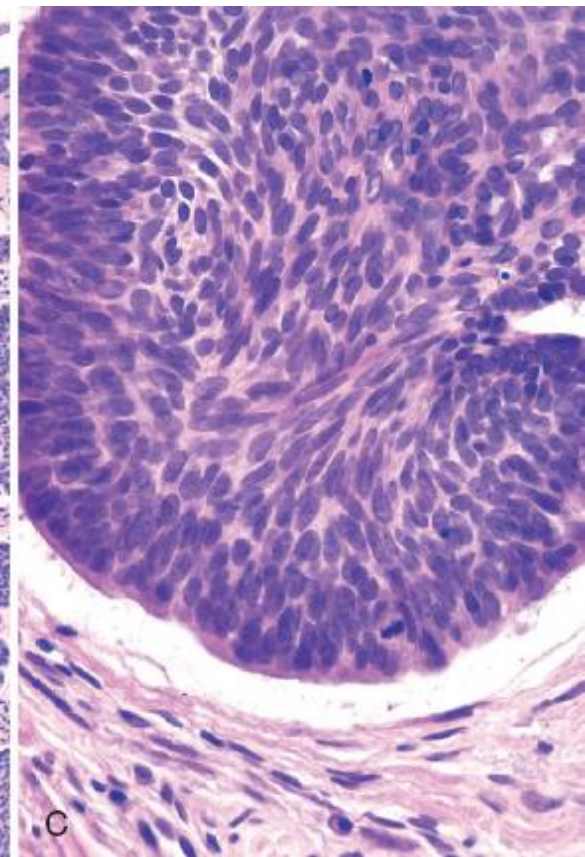
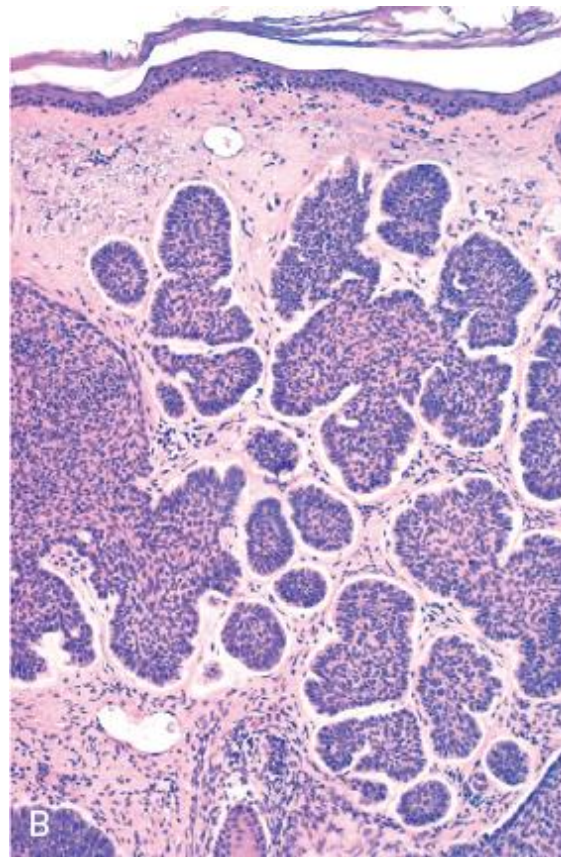
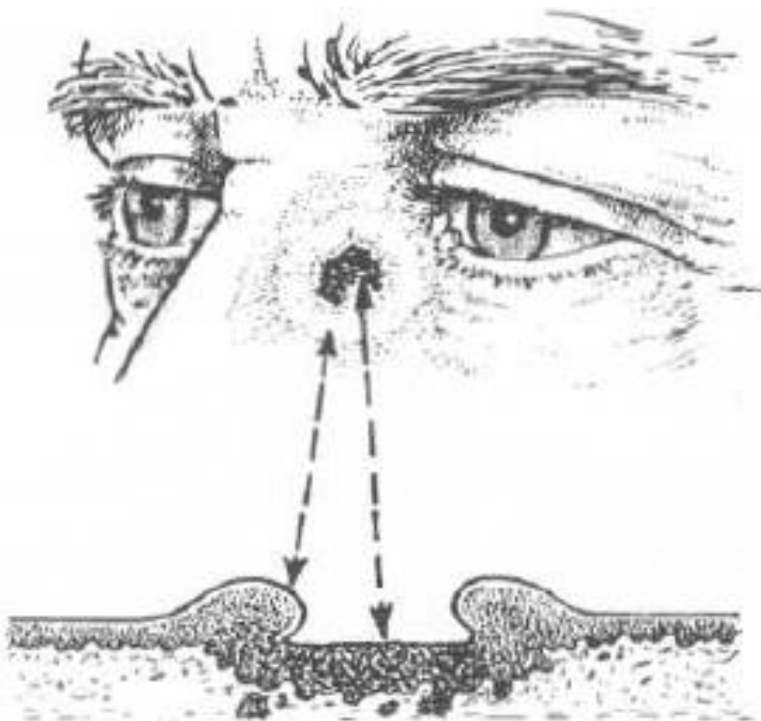
- Jinak – berkapsul/ simpai, berbatas tegas
- Ganas – tidak berkapsul, difus (tidak berbatas tegas)



Carcinoma Mammae VS FAM ???

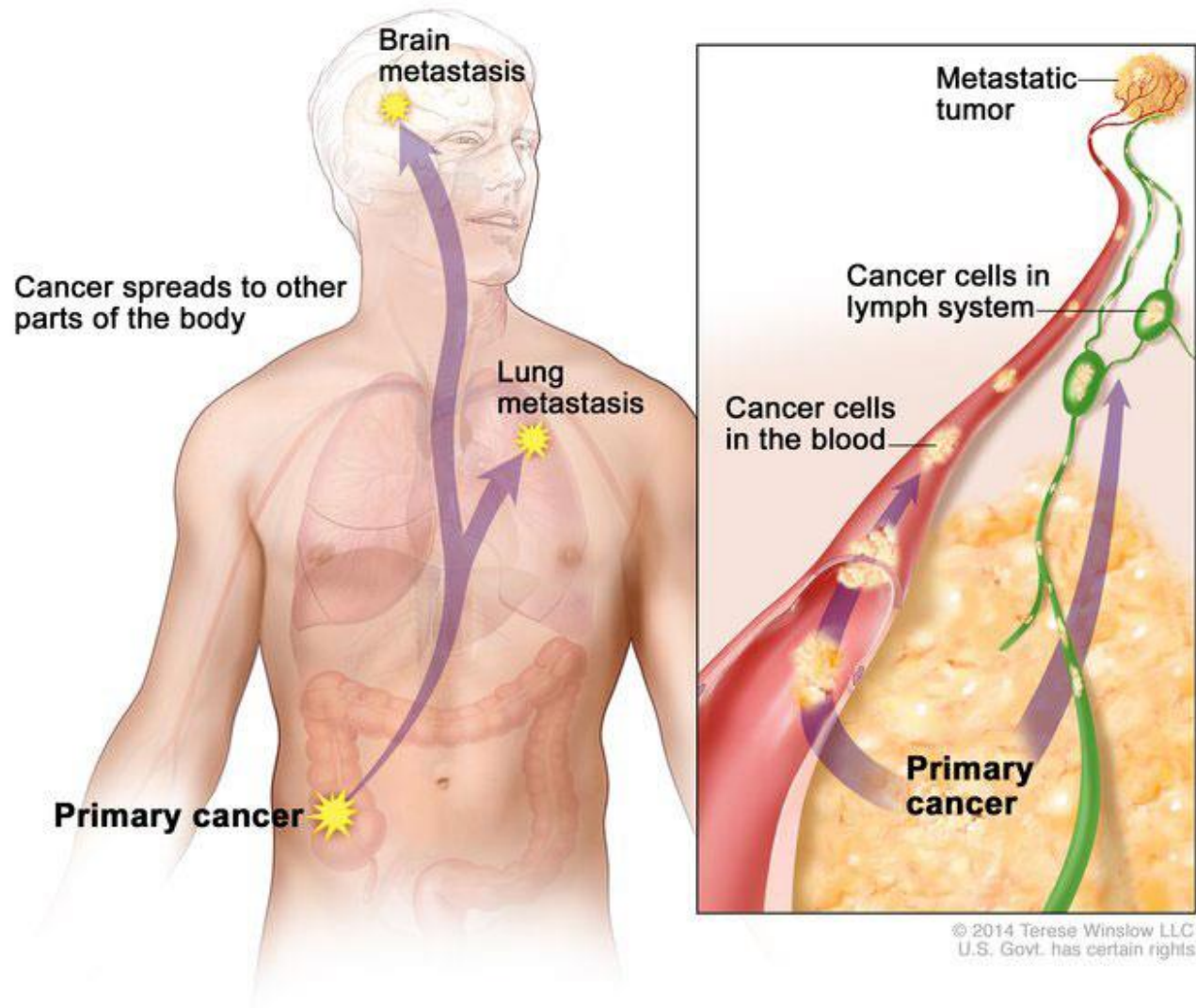
Metastasis

- Menyebar jauh ke organ lain dari lokasi primer
- Tidak semua kanker
- Karsinoma Sel Basal (KSB) dan kanker otak (astrocitoma) – destruksi lokal namun meta (-)



Karsinoma sel basal – rodent's ulcer

Metastasis



Karsinogenesis

- *Genetic damage* – mutasi
- Gen-gen regulator normal
 1. Growth promoting – proto-oncogen
 2. Growth – inhibiting tumor suppresor gene
 3. Gen yg meregulasi kematian sel terprogram
 4. Gen terlibat perbaikan DNA

Karsinogenesis

Oncogene

- Gen yg menginduksi transformasi fenotip ketika terekspresi.
- Mutasi/ overekspresi – proto-oncogene
- Faktor transkripsi, *growth regulating, cell survival*

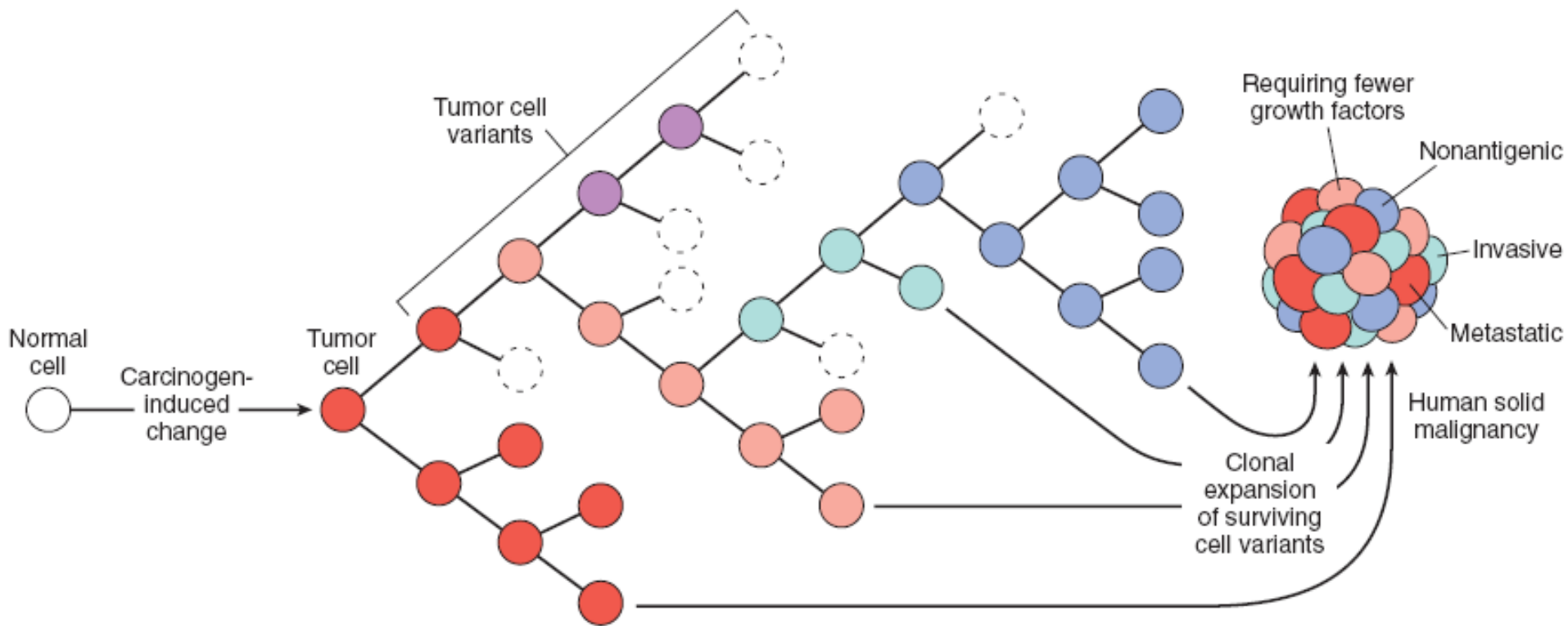
Karsinogenesis

Tumor suppressor gene

- Normal mencegah pertumbuhan berlebihan
- Governor – Rb → mutasi – “rem” proliferasi sel hilang → Retinoblastoma
- Guardian – TP53 → Ca ovarium, Ca.....

Karsinogenesis

- Multistep



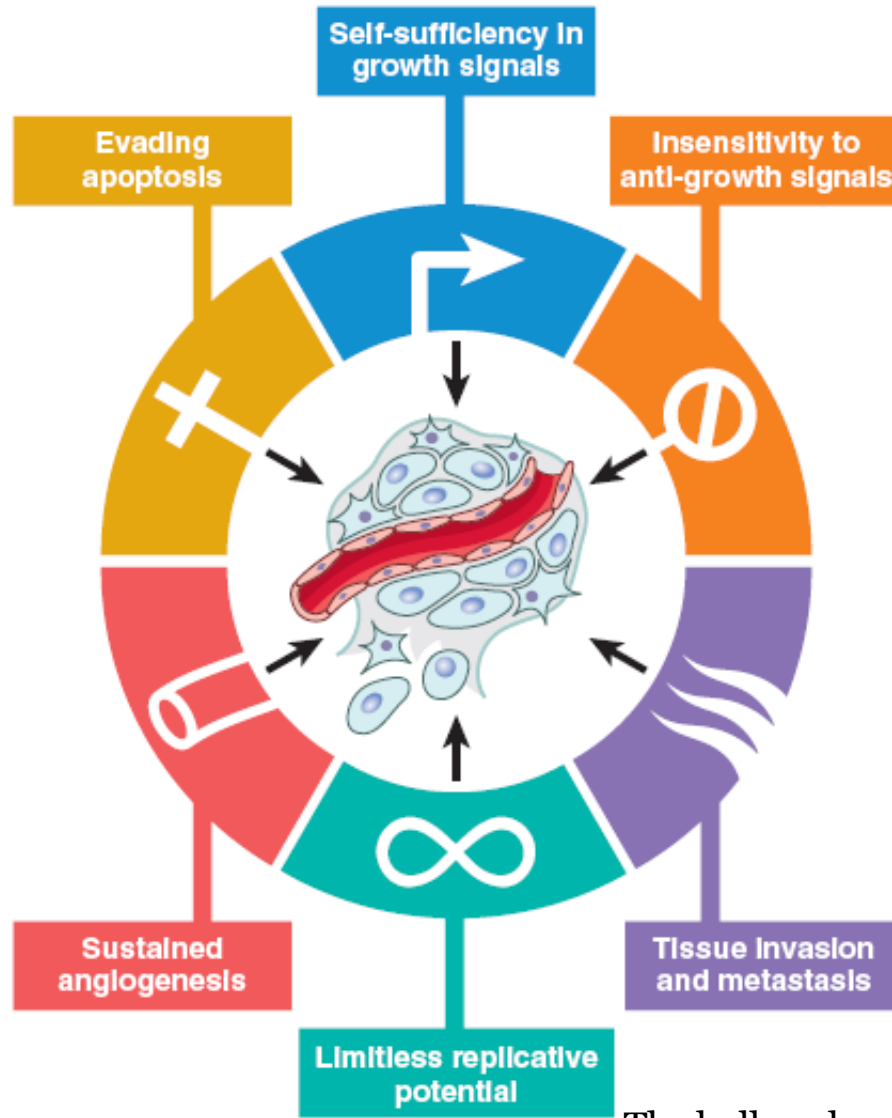
TRANSFORMATION

PROGRESSION

PROLIFERATION
OF GENETICALLY
UNSTABLE CELLS

TUMOR CELL
VARIANTS:
HETEROGENEITY

Hallmarks kanker



The hallmarks of cancer. Cell. 2000. Hanahan.

The hallmarks of cancer

- 1. Swasembada sinyal pertumbuhan:** sel kanker mendapatkan dorongan mandiri untuk menyebar, mitosis patologis, dengan aktivasi onkogen (ras atau myc).
- 2. Tak peka sinyal penghambat pertumbuhan (anti pertumbuhan).** Sel kanker menonaktifkan gen penekan tumor, seperti retinoblastoma (Rb) yang normalnya menghambat pertumbuhan
- 3. Menghindari kematian sel terprogram (apoptosis),** sel kanker menekan dan menonaktifkan gen dan jalur yang normalnya membuat sel bisa mati

The hallmarks of cancer

4. **Potensi replikasi tak terbatas:** sel kanker mengaktifkan jalur gen khusus yang membuatnya *abadi*.
5. **Angiogenesis berkelanjutan:** sel kanker mendapat kapasitas menarik pasokan darah sendiri dan pembuluh darah angiogenesis tumor.
6. **Invasi jaringan tubuh dan metastasis:** sel kanker mendapat kapasitas untuk berpindah ke organ lain, menginvasi jaringan lain.

Etiologi

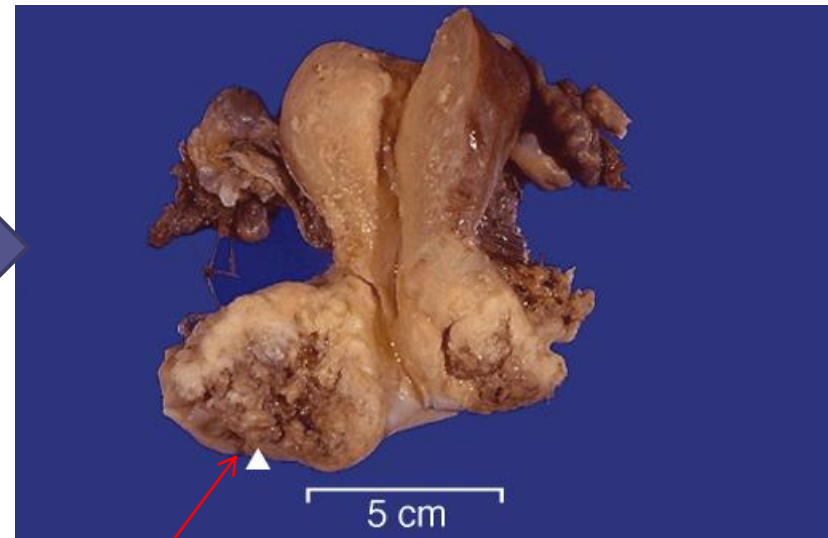
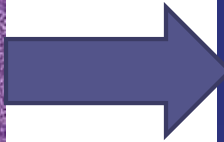
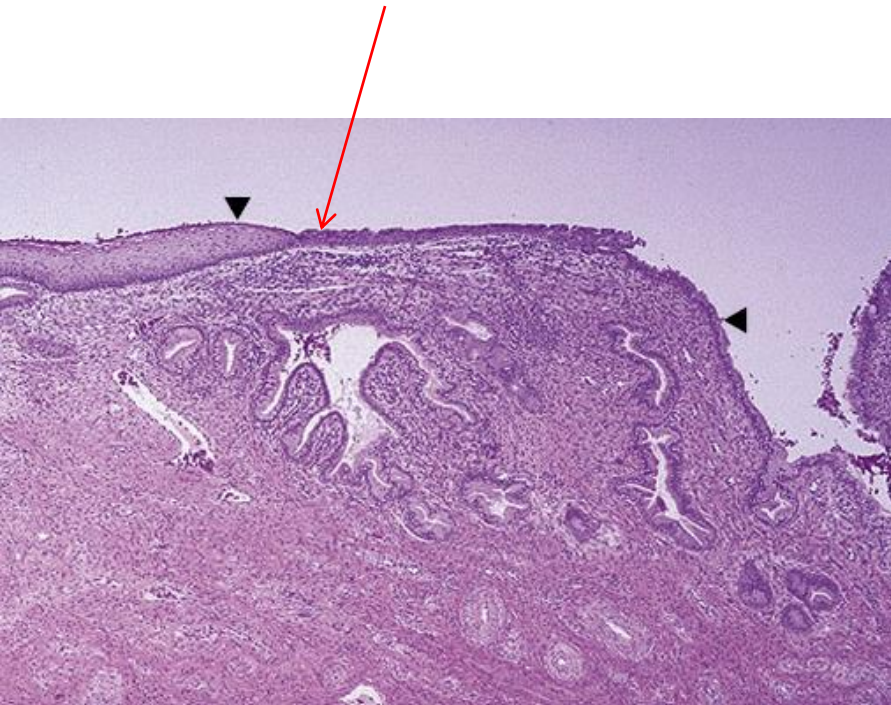
Agen karsinogenik:

1. Bahan kimia
2. Radiasi
3. Mikroba

Viral dan Mikroba onkogenesis

- HPV (human papilloma virus) – cervical cancer
- EBV (Epstein- Barr virus) – Ca nasofaring, limfoma
- HBV (Hepatitis B virus) – Hepatocellular carcinoma
- Helicobacter Pylori – adenocarcinoma gaster

HPV high risk (16,18)



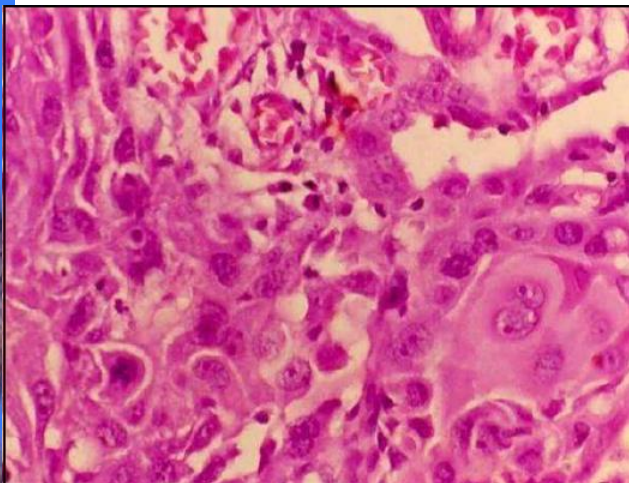
Karsinoma sel skuamosa serviks

Aspek klinik

- Kaheksia – wasting
- Sindrom paraneoplastik – kanker paru
- Derajat dan stadium kanker – klinis dan patologis

Diagnosis

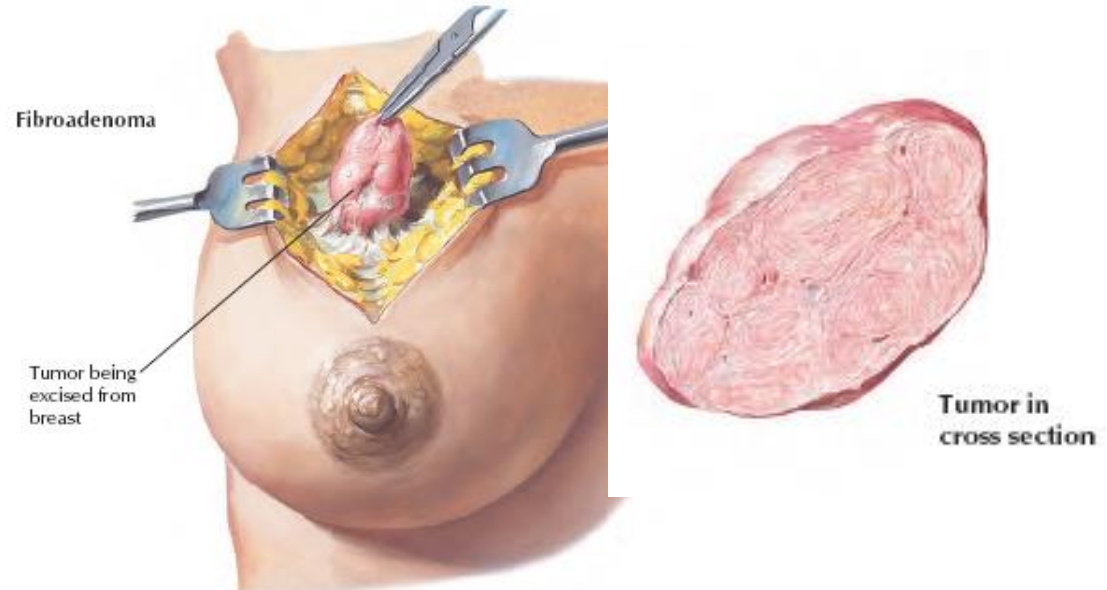
- Klinis
- Radiologis
- Diagnosis Patologik – diagnosis potong beku (*Frozen Section*)
- Spesimen: biopsi, eksisi, aspirasi jarum halus, smear (PAP smear)
- Tumor marker: CA 125, AFP, PSA
- Molekular – mutasi – target terapi



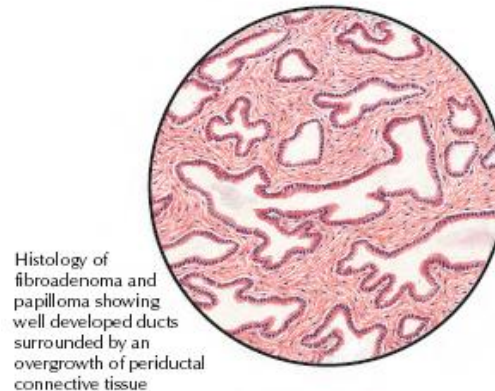
RO thorax: Kanker paru jenis non-small cell

Patolog → menegakkan diagnosa → Th/ (klinikus)

- Makroskopik →



- Mikroskopik →



Terapi

- Operasi/ pembedahan
- Kemo - Radiasi
- Hormonal
- Target terapi





Universitas Kristen Indonesia

Fakultas Kedokteran

SURAT KEPUTUSAN
No. : 069/UKI.F5.D/HKP.3.5.6/2021
tentang

PENUGASAN TENAGA AKADEMIK DALAM MEMBERIKAN KULIAH PAKAR PIMPINAN FAKULTAS KEDOKTERAN UNIVERSITAS KRISTEN INDONESIA

MENIMBANG : Bahwa untuk kelancaran proses belajar mengajar dan meningkatkan mutu pendidid di FKUKI diperlukan penugasan tenaga akademik FKUKI untuk memberi Kuliah Pakar

MENGINGAT : 1. Peraturan Pemerintah No. 60 tahun 1999 tentang Pendidikan Tinggi
2. Surat Keputusan Dekan FKUKI No. 53/SK/FKUKI/11.2006 tanggal November 2006 tentang Pemberlakuan Kurikulum Berbasis Kompetensi (KBK FKUKI
3. Surat Keputusan Rektor UKI No. 90/UKI.R/SK/SDM.8/2018 tent pengangkatan Dekan Fakultas Kedokteran UKI
4. Surat keputusan pengangkatan sebagai tenaga akademik

MEMUTUSKAN

MENETAPKAN : 1. Penugasan dalam memberikan Kuliah Pakar :
Nama dr. Fajar L. Gultom, Sp.PA
Departemen Patologi Anatomi
Blok 5 (Biomedik)
Judul Materi Hemodinamik 2
Semester genap 2020/2021
Kelas A : 0,21 SKS
B : 0,21 SKS
SKS 0,42 SKS
2. Apabila dikemudian hari ternyata terdapat kekeliruan dalam Surat Keputu ini akan diperbaiki sebagaimana mestinya

Asli Surat Keputusan ini disampaikan kepada yang bersangkutan untuk diketahui

Ditetapkan di : Jakarta
Pada tanggal : 15 April 2021
Dekan,

Dr. dr. Robert Hotman Sirait, Sp.An.
NIP. UKI. 031 545

Tembusan:

1. Rektor UKI
2. Wakil Dekan Bidang Akademik FKUKI



Proses keseimbangan cairan, elektrolit dan asam-basa

Fajar L. Gultom
Departemen Patologi Anatomik
FK UKI
2021

Pendahuluan

- Tubuh manusia terdiri dari 60% air
- Cairan tubuh:
 1. Di dalam sel (intraseluler)
 2. Di luar sel (ekstraseluler) → cairan intravaskular dan interstitial (cairan limfe dan cairan serebrospinal)

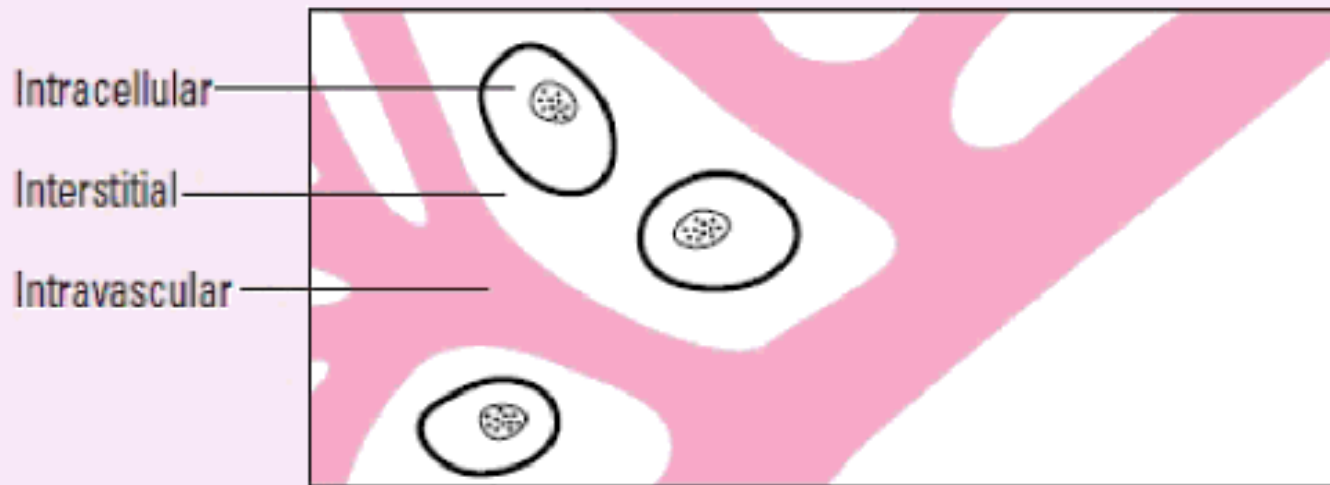
Jumlah cairan masuk = jumlah cairan keluar

→ Keseimbangan / Homeostasis

- The health of cells and tissues depends on circulation of blood (oxygen, nutrients, removes waste)
- Three major components of the Cardiovascular system: the heart, the blood vessels and the blood
- Blood: water, salts, proteins, elements regulate clotting (coagulation factors and platelets), red cells, white cells
- Disorder of hemodynamics: edema, effusions, congestion, shocks
- Disorder of abnormal bleeding, clotting (thrombosis), embolism

Fluid compartments

The primary fluid compartments in the body are intracellular and extracellular. Extracellular is further divided into interstitial and intravascular. Capillary walls and cell membranes separate intracellular fluids from extracellular fluids.



Daily total intake 2,600 ml

- Liquids 1,500 ml
- Solid foods 800 ml
- Water of oxidation 300 ml

Daily total output 2,600 ml

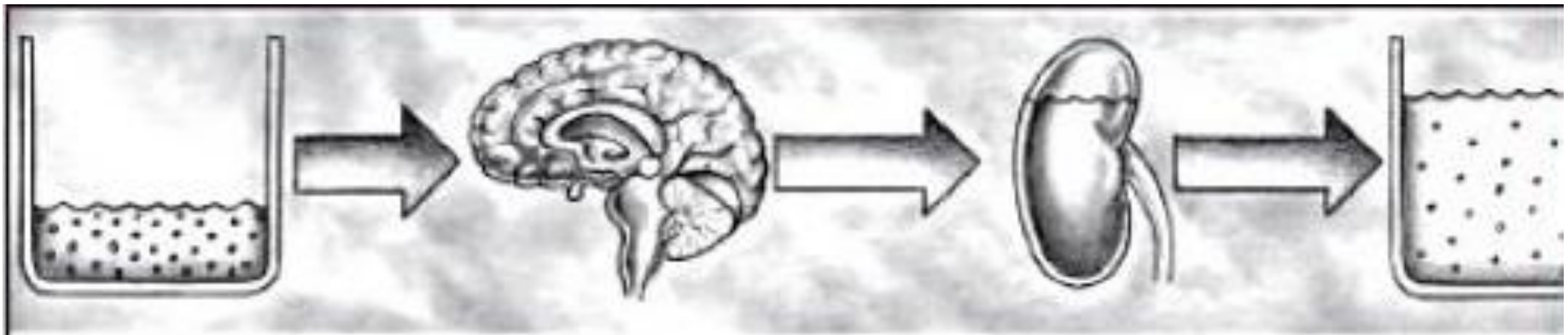
- Skin 600 ml
- Lungs 400 ml
- Kidneys (urine) 1,500 ml
- Intestines (stool) 100 ml

Pendahuluan

Faktor- faktor yang mempengaruhi keseimbangan cairan:

1. Tekanan hidrostatik kapiler
2. Tekanan osmotik koloid
3. Permeabilitas kapiler
4. Kadar ion Na^{2+}

Pengaturan keseimbangan elektrolit dan cairan tubuh oleh ginjal → dipengaruhi hormon ADH (anti diuresis hormon) dan aldosteron

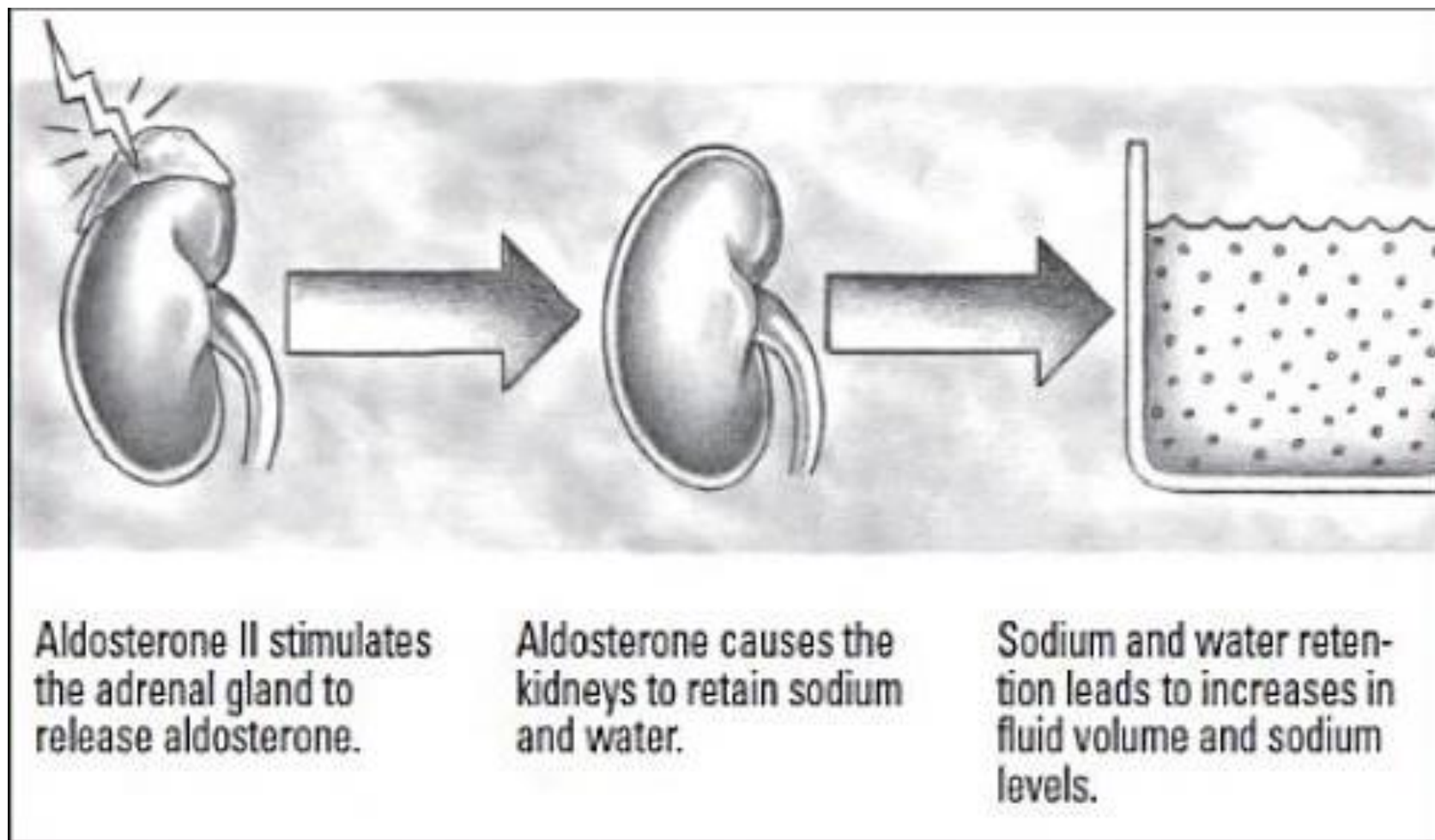


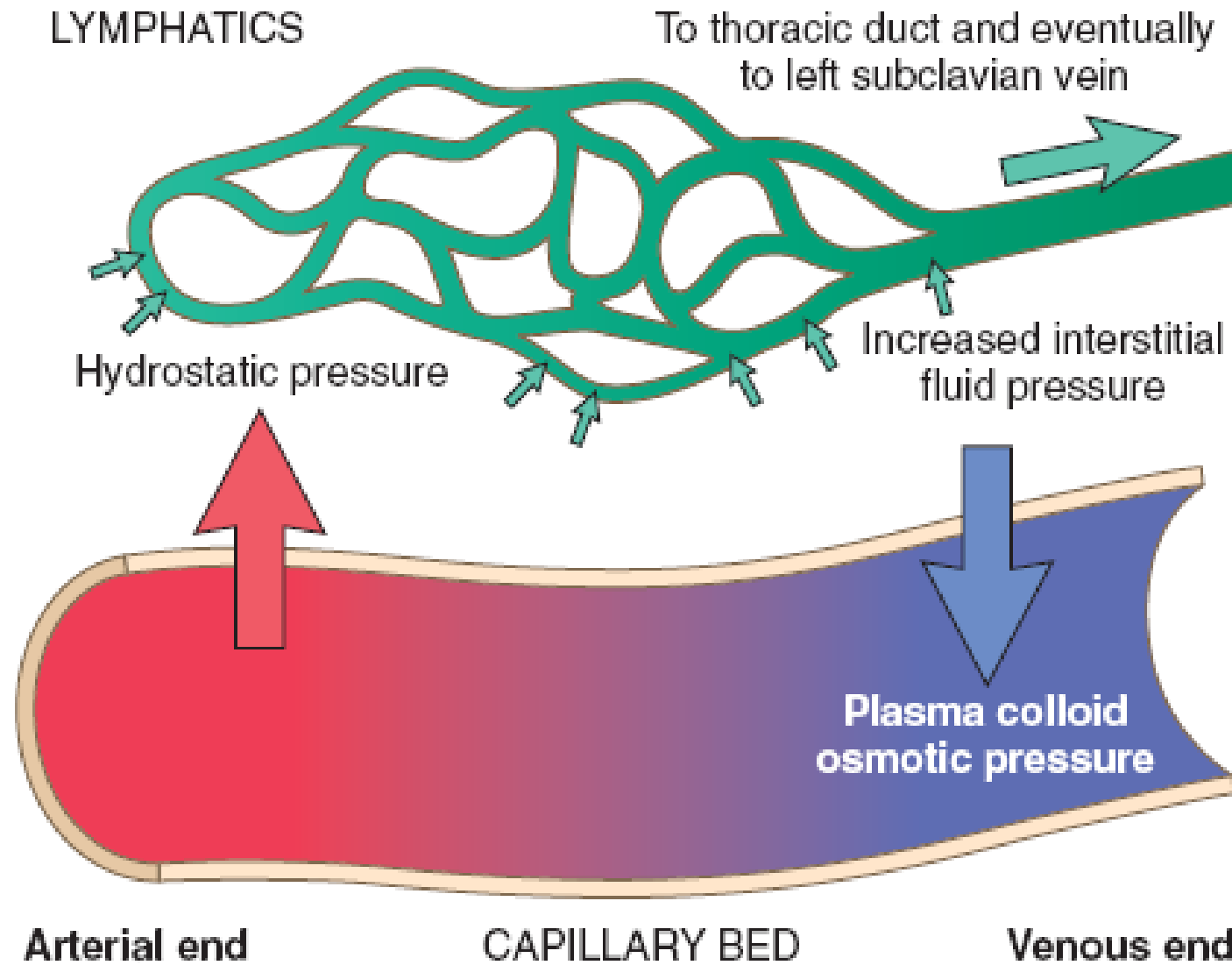
The hypothalamus senses low blood volume and increased serum osmolality and signals the pituitary gland.

The pituitary gland secretes ADH into the bloodstream.

ADH causes the kidneys to retain water.

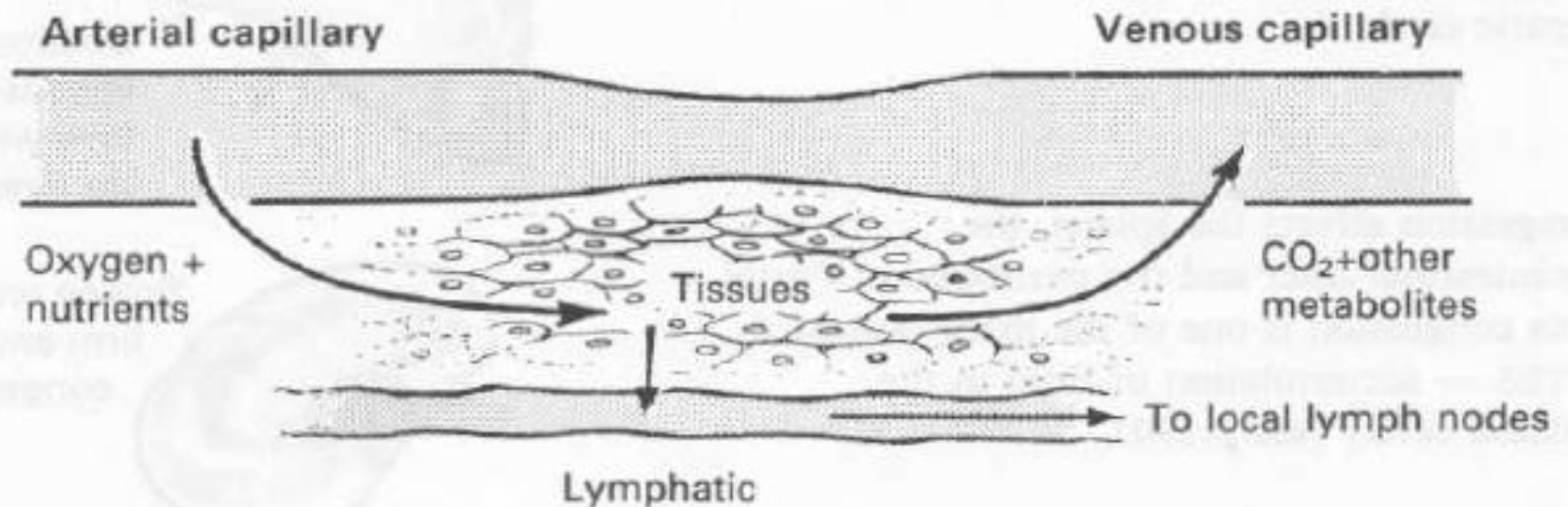
Water retention boosts blood volume and decreases serum osmolality.





NORMAL TISSUE FLUID CIRCULATION

There is a continuous interchange of fluid between blood and tissues. Some fluid enters the lymphatics before eventually returning to the blood stream.



Arterial capillary

BP (35 mmHg) — OP(25 mmHg)
∴ Filtration pressure = 10mmHg

Venous capillary

OP (25mmHg) — BP (10mmHg)
∴ Osmotic attraction = 15mmHg

Tissues

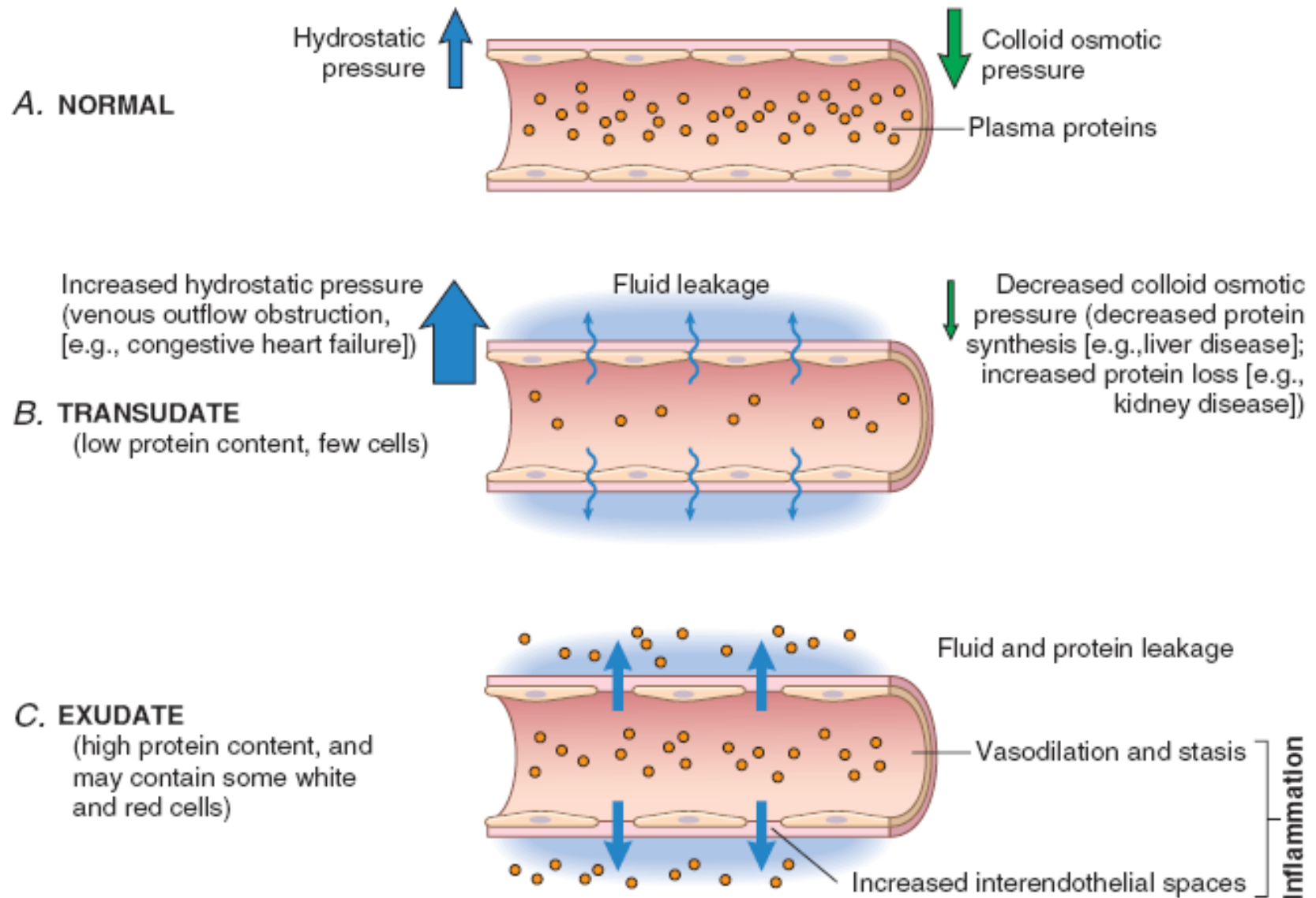
A schematic diagram of a blood vessel segment showing the transition from an arterial capillary to a venous capillary. The vessel is represented by a horizontal tube with a wavy line indicating its lumen. Below the vessel, a cluster of cells represents the tissues. Arrows indicate the direction of fluid movement: one arrow points from the arterial end of the capillary into the tissue space, and another arrow points from the tissue space into the venous end of the capillary. The diagram is divided into three horizontal sections: the top section for the arterial capillary, the middle section for the tissues, and the bottom section for the venous capillary. The top section contains text about blood pressure (BP) and oncotic pressure (OP) at the arterial end, resulting in a filtration pressure. The bottom section contains text about OP and BP at the venous end, resulting in an osmotic attraction. The middle section is labeled 'Tissues' and shows a cluster of cells.

Edema

- Pengumpulan cairan berlebihan pada sela-sela jaringan atau rongga tubuh
- Cairan edema dikelompokkan:
 1. Peradangan/**eksudat**
Kadar protein tinggi dan BJ > 1,20
 2. Non peradangan/**transudat**
Kadar protein rendah dan BJ < 1,15
- Umum → anasarka
- Pada rongga serosa → hidrothoraks (efusi pleura), hidroperikardium dan hydroperitoneum (asites)

Edema

- Penyebab edema non radang:
 1. Peningkatan tekanan hidrostatik
 2. Penurunan tekanan osmotik koloid
 3. Obstruksi saluran limfe
- Penyebab edema radang → permeabilitas kapiler ↑ (sitokin/ endotoksin)
- Penyebab lain → gangguan pertukaran natrium/keseimbangan elektrolit





Pitting edema vs non-pitting edema

<http://en.wikipedia.org/wiki/Edema>

Peningkatan tekanan hidrostatik

- Tekanan darah **mendorong** cairan **dari** pembuluh darah **ke** arah rongga interstitial.
- Edema terjadi:
 1. Tekanan vena sentral meningkat → darah balik vena perifer ke ventrikel terhambat
 2. Stasis darah venula dan kapiler → peningkatan tekanan intrakapiler → mendorong cairan ke rongga interstitial

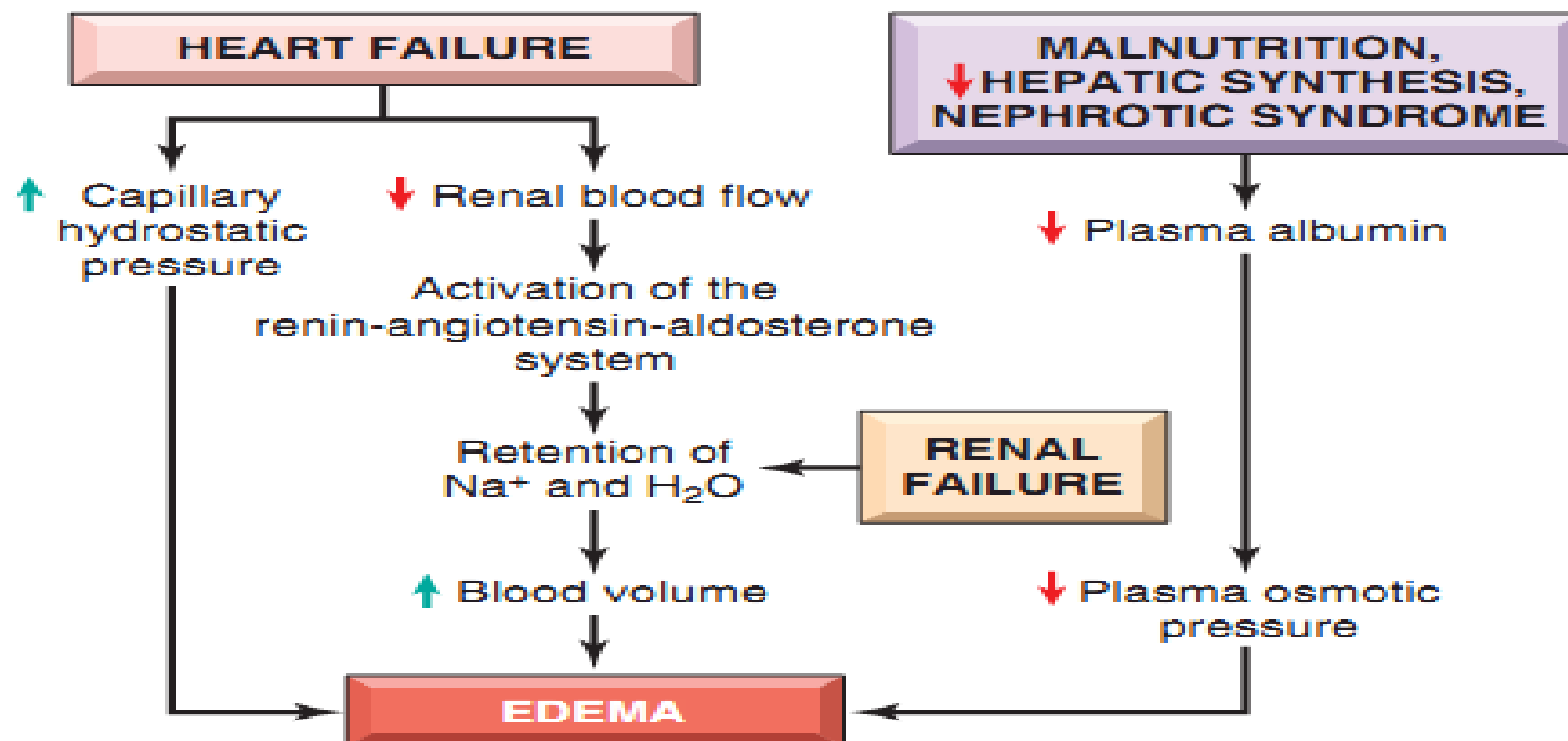


Figure 3–3 Pathways leading to systemic edema due to heart failure, renal failure, or reduced plasma osmotic pressure.

Penurunan tekanan osmotik

- Tekanan osmotik koloid → **mempertahankan** cairan di pembuluh darah agar tidak mengalir ke rongga interstitial (albumin)
- Albumin dihasilkan di hati
- Penurunan kadar albumin terjadi pada:
 1. Kerusakan hati
 2. Sindroma nefrotik
 3. Malnutrisi

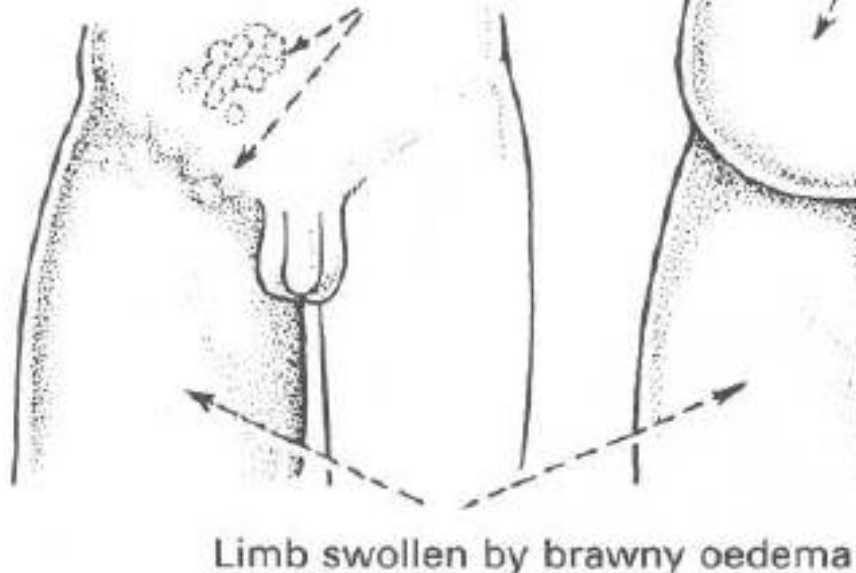


<http://en.wikipedia.org/wiki/Ascites>

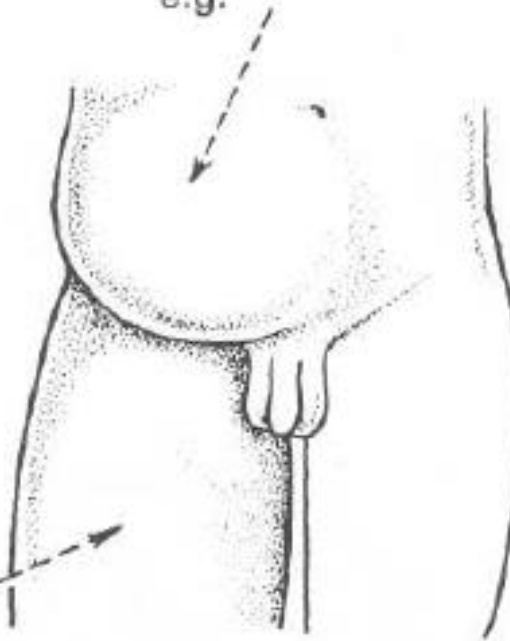
Obstruksi aliran limfe

- Pembuluh limfe berfungsi sebagai jalan utama aliran cairan interstitial dan cairan limfe.
- Obstruksi → edema pada daerah distal obstruksi
- Contoh: kanker payudara, fibrosis pascaradiasi, filariasis

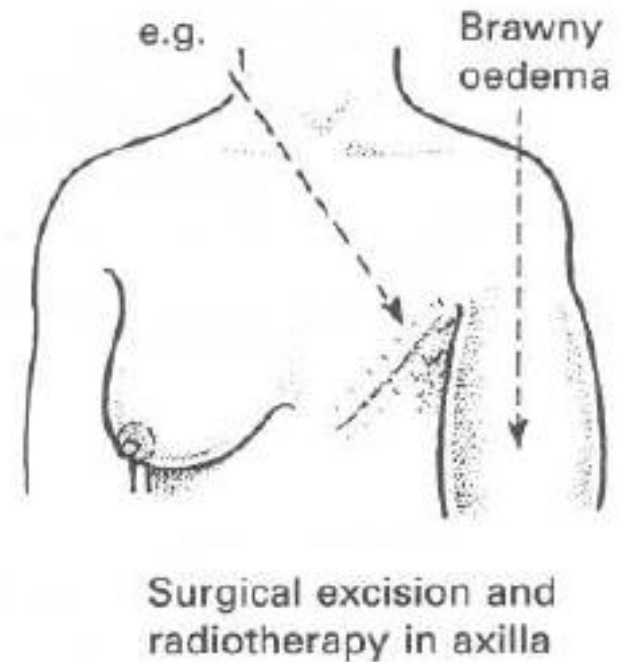
1. Obstruction by tumour permeation of lymphatics and/or destruction of lymph nodes, e.g. pelvic and groin nodes.



2. Direct pressure by large tumour mass.
e.g.

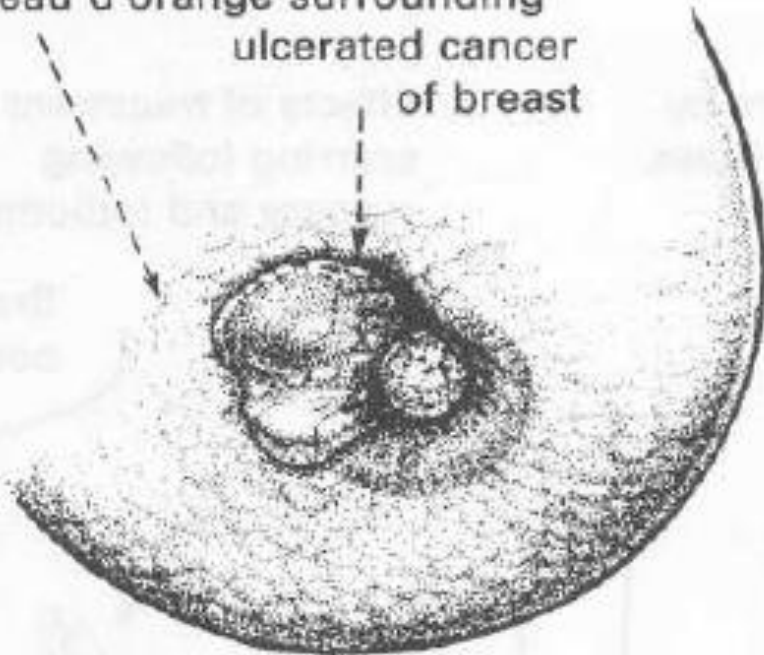


3. Effects of treatment — scarring following surgery and radiotherapy.

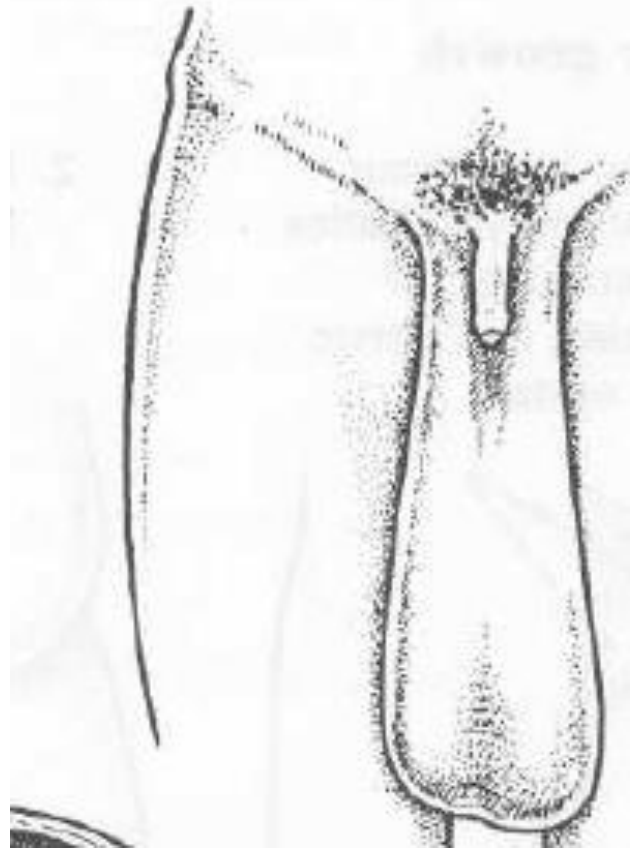


Peau d'orange — where the lymphatics of the skin are blocked by tumour.

Peau d'orange surrounding
ulcerated cancer
of breast



Elephantiasis — where a limb and/or the scrotum is massively enlarged due to filarial lymphatic blockage.



Peningkatan permeabilitas pembuluh darah

- Permeabilitas pembuluh darah menjaga agar protein plasma tetap berada dalam pembuluh darah kapiler
- Permeabilitas pembuluh darah ↑:
 1. Peradangan → sekresi sitokin
 2. Infeksi bakteri → produksi endotoksin
 3. Syok anafilaktik → reaksi antigen dan Ig E pada sel mast → histamin
- Permeabilitas kapiler ↑ protein plasma keluar ke jaringan interstitial

Gangguan pertukaran natrium

- Gangguan pertukaran Na^{2+} → hipertoni → menahan air yg ada di pembuluh darah atau ruang interstitial
- Berperan pada edema akibat:
 1. Payah jantung → peningkatan aldosteron
 2. Sirosis hepatis → asites → tekanan vena porta meningkat → aktivasi renin angiotensin aldosteron → retensi Na^{2+} dan ekskresi K^+
 3. Penyakit ginjal akut → retensi Na^{2+}
- *Pitting* edema pada ekstremitas bawah



Elektrolit

Intraselular dan ekstraselular

Ekstraselular:

- Sodium → interaksi sel saraf dan sel otot
- Klorida → menjaga tekanan osmotik, sel mukosa gaster → asam lambung
- Kalsium → stabilisasi membran sel, mengurangi permeabilitas, kontraksi otot, membentuk tulang dan gigi
- Bikarbonat → keseimbangan asam-basa

Elektrolit

Intraselular

- Potasium → kontraksi otot, konduksi saraf, responsif membran miokardial
- Fosfat → metabolisme energi
- Magnesium → reaksi enzim, kontraksi neuromuskular, sistem saraf dan kardiovaskular

Normal electrolyte levels

To maintain homeostasis, the body keeps electrolytes within a normal range, as shown below. All normal electrolyte levels are measured in serum.

Electrolyte	Normal levels
Sodium	135 to 145 mEq/L (SI, 135 to 145 mmol/L)
Potassium	3.5 to 5 mEq/L (SI, 3.5 to 5 mmol/L)
Calcium, total	8.2 to 10.2 mg/dl (SI, 2.05 to 2.54 mmol/L)
Calcium, ionized	4.65 to 5.28 mg/dl (SI, 1.1 to 1.25 mmol/L)
Phosphates	2.7 to 4.5 mg/dl (SI, 0.87 to 1.45 mmol/L)
Magnesium	1.3 to 2.1 mEq/L (SI, 1.3 to 2.1 mmol/L)
Chloride	100 to 108 mEq/L (SI, 100 to 108 mmol/L)

Elektrolit

Faktor-faktor yang mempengaruhi:

- Input dan output cairan
- Keseimbangan asam-basa
- Sekresi hormon (aldosteron)
- Fungsi sel normal

Kadar elektrolit yang diukur → ekstraseluler

Gangguan elektrolit

Sodium

- Hiponatremia – hipernatremia
- Pasien rawat inap, lansia/ geriatri
- Ringan → asimtomatik, *self limiting*
- Berat → morbiditas & mortalitas

Box 1: Classification of hyponatraemia

Hypovolaemia

Extrarenal loss, urine sodium <30 mmol/l

- Dermal losses, such as burns, sweating
- Gastrointestinal losses, such as vomiting, diarrhoea
- Pancreatitis

Renal loss, urine sodium >30 mmol/l

- Diuretics
- Salt wasting nephropathy
- Cerebral salt wasting
- Mineralocorticoid deficiency (Addison's disease)

Hypervolaemia*

Urine sodium <30 mmol/l

- Congestive cardiac failure
- Cirrhosis with ascites
- Nephrotic syndrome

Urine sodium >30 mmol/l

- Chronic renal failure

Euvolaemia

Urine sodium >30 mmol/l

- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)†
- Hypothyroidism
- Hypopituitarism (glucocorticoid deficiency)
- Water intoxication:
 - Primary polydipsia
 - Excessive administration of parenteral hypotonic fluids
 - Post-transurethral prostatectomy

Box 3: Classification of hypernatraemia

Hypovolaemia

- Dermal losses—for example, burns, sweating
- Gastrointestinal losses—for example, vomiting, diarrhoea, fistulas
- Diuretics
- Postobstruction
- Acute and chronic renal disease
- Hyperosmolar non-ketotic coma*

Hypervolaemia

- Iatrogenic (hypertonic saline, tube feedings, antibiotics containing sodium, or hypertonic dialysis)
- Hyperaldosteronism†

Euvolaemia

- Diabetes insipidus (central, nephrogenic, or gestational)
- Hypodipsia
- Fever
- Hyperventilation
- Mechanical ventilation

*Sodium often raised, even after correction for glucose

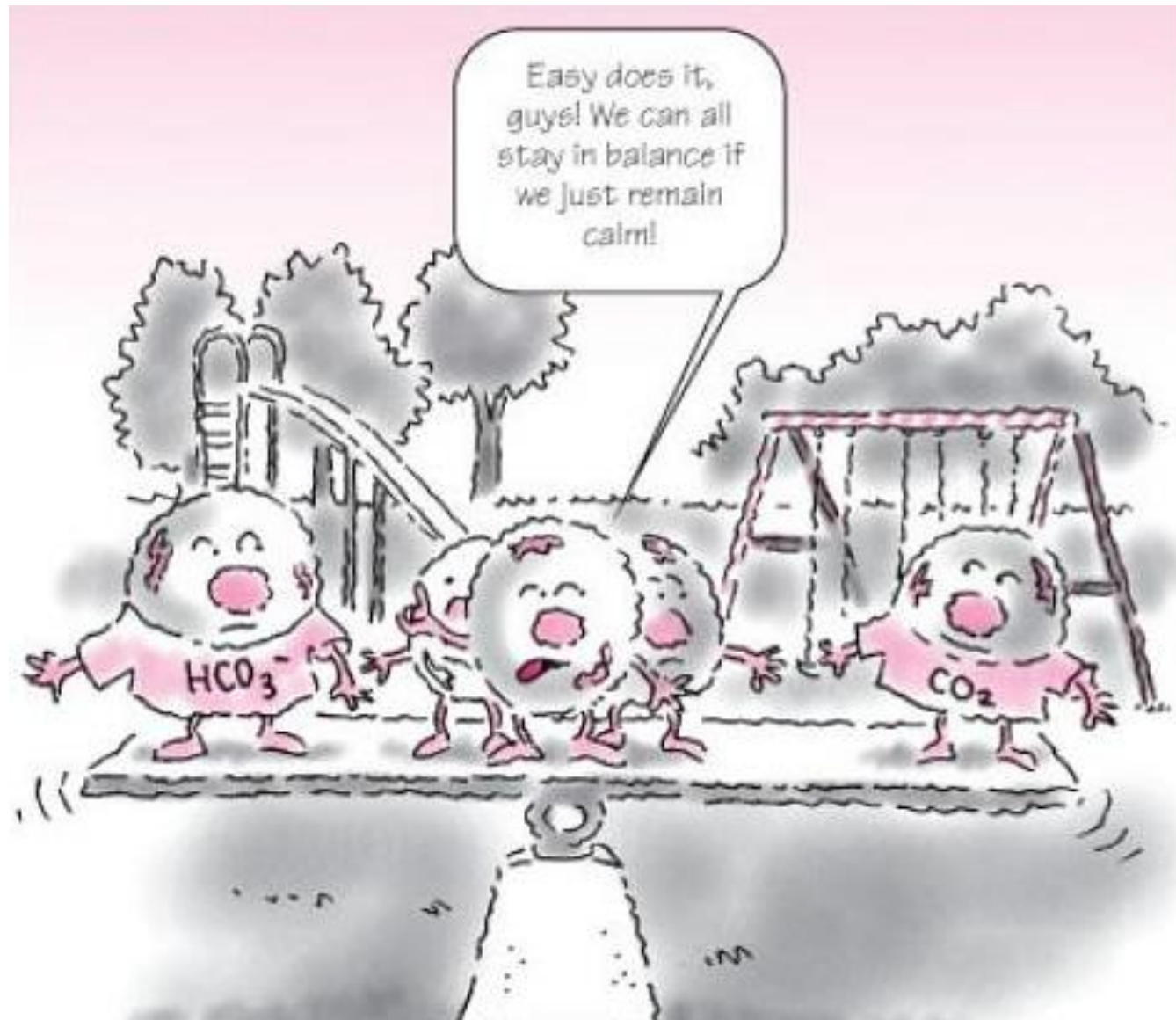
†Typically mildly elevated sodium ~147 mmol/L, so rarely a clinical problem

Gangguan elektrolit

Kalium

- Hipokalemia – hiperkalemia
- Mengancam nyawa → emergensi

Asidosis & Alkalosis



Asidosis

Peningkatan sistemik konsentrasi ion hidrogen (H^+), penyebab:

- Paru-paru gagal dalam eliminasi CO_2
- Akumulasi produk asam dari metabolisme (asam laktat)
- Konsentrasi H^+ \uparrow
- Diare persisten $\rightarrow HCO_3^- \downarrow$
- Gagal ginjal \rightarrow reabsorpsi HCO_3^- & sekresi H^+

- pH normal: 7,35-7,45
- Asidosis \rightarrow pH < 7,35
Akumulasi H^+ atau deplesi basa
- Alkalosis \rightarrow pH > 7,45
Akumulasi HCO_3^- atau deplesi asam

Alkalosis

Penurunan sistemik konsentrasi H^+ , penyebab:

- Hiperventilasi $\rightarrow CO_2 \downarrow$
- Muntah $\rightarrow H^+ \downarrow$
- Asupan basa \uparrow